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Important Books Received

Physiological chemistry; textbook and manual for students. By Albert P. Mathews, Ph.D., Professor of Biochemistry, University of Cincinnati. *Fourth Edition.* 1925: pp. 1233; 7¼ x 4¾ in.; 109 illustrations; $6.50. William Wood and Co., New York City.

Bacterial infection, with special reference to dental practice. By J. L. T. Appleton, Jr., B.S., D.D.S., Professor of Microbiology and Bacteriopathology, Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania. 1925: pp. 474; 7¼ x 4 in.; 96 illustrations; $6.00. Lea and Febiger, Philadelphia.

Dental materia medica and therapeutics, with special reference to the rational application of remedial measures to dental diseases. By Hermann Prinz, A.M., D.D.S., M.D., Sc.D., Professor of Materia Medica and Therapeutics, Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania. *Sixth edition.* 1926: pp. 632; 7 x 4½ in.; 146 illustrations; $6.00. C. V. Mosby Co., St. Louis.

Technic of oral radiography. By Clarence O. Simpson, M.D., D.D.S., F.A.C.D. 1926: pp. 207; 6¾ x 4 inches; 165 illustrations; $5.00. C. V. Mosby Co., St. Louis.

Orthodontics: an historical review of its origin and evolution. *Two volumes.* By Bernhard Wolf Weinberger, D.D.S., Librarian of First District Dental Society of the State of New York, and Advisory Librarian, Dental Department, New York Academy of Medicine, New York City. 1926: pp. 1011; 7¼ x 4¾ in.; 599 illustrations; $25.00 (*two volumes*). C. V. Mosby Co., St. Louis.

American text-book of operative dentistry. By Marcus L. Ward, D.D.S., D.D.Sc., F.A.C.D., Editor; Professor of Dental Metallurgy and Crown and Bridge Work, and Dean, College of Dental Surgery, University of Michigan. *Sixth edition.* 1926: pp. 818; 7¼ x 4½ in.; 783 illustrations; $10.00. Lea and Febiger, Philadelphia and New York.

Annuario dental 1926. By Salvador de Casas (and numerous collaborators). *Fifth edition.* 1926: pp. 462; 6¾ x 4½ in.; nearly 200 illustrations; 12 Pesetas. Salvador de Casas, Calle de Galileo, No. 5, Madrid, Spain.

A textbook of exodontia—exodontia, oral surgery, and anesthesia. Leo Winter, D.D.S., Professor of Oral Surgery, College of Dentistry, New York University. 1927: pp. 364; 7¼ x 4½ in.; 329 illustrations; $7.50. C. V. Mosby Co., St. Louis.

Harelip and cleft palate (cheiloschisis, uranoschisis, and staphyloschisis). By Matthew N. Federspiel, B.Sc., D.D.S., M.D., F.A.C.S., Professor of Oral Surgery, Marquette University. 1927: pp. 200; 6¾ x 4 in.; 156 illustrations; $5.00. C. V. Mosby Co., St. Louis.

A text-book of bacteriology for dental students. By Arthur Bulleid, L.R.C.P. (London), M.R.C.S., L.D.S. (England), Assistant Dental Surgeon and Assistant Bacteriologist, Guy's Hospital, London. 1927: pp. 219; 6¾ x 4 in.; 60 illustrations; $4.50. C. V. Mosby Co., St. Louis.

Textbook of biological chemistry. By James B. Sumner, Ph.D., Assistant Professor of Biological Chemistry, Cornell University (Ithaca). 1927: pp. 283; 6¾ x 4 in.; 6 illustrations; $3.50. MacMillan Co., New York City.

The contributions of the Eastern Association of Graduates of the Angle School of Orthodontia to Orthodontics. A review and index of its proceedings. By Bernhard Wolf Weinberger, D.D.S. 1927: pp. 48; 7¼ x 4½ in.; no illustrations. Eastern Association of Graduates of the Angle School of Orthodontia.

Gangrene of the dental pulp and its treatment, as based on a biochemical investigation. By Gustav Hedstrom (translated by Carl Westman). 1927: pp. 178; 6¾ x 4 in.; 35 figures. Dental-aktiebolaget, Stockholm, Sweden.

Lectures [6] on the biologic aspects of colloid and physiologic chemistry. A series of lectures given at the Mayo Foundation and the Universities of Minnesota, Iowa, Washington (St. Louis), and the Des Moines Academy of Medicine, Iowa. By six investigators in the field. Introduction by Louis B. Wilson, M.D., Director of the Mayo Foundation for Medical Education and Research. 1927: pp. 244; 6¾ x 3½ in.; 85 illustrations; $2.50. W. B. Saunders Company, Philadelphia and London.

American illustrated medical dictionary. By W. A. Newman Dorland, A.M., M.D., F.A.C.S., member of the Committee on Nomenclature and Classification of Diseases, American Medical Association, and Editor of the "American Pocket Medical Dictionary;" with the collaboration of E. C. L. Miller, M.D., Professor of Bacteriology and Biochemistry, Medical College of Virginia. *Fourteenth edition.* 1927: pp. 1388; 8¼ x 4¾ in.; 319 illustrations; flexible binding—plain, $7.00; thumb index, $7.50. W. B. Saunders Company, Philadelphia and London.

Dental education in the United States and Canada. By William J. Gies, M.S., Ph.D., Sc.D., LL.D., F.A.C.D., Professor of Biological Chemistry, Columbia University; Executive officer of the Board of Editors, *Journal of Dental Research;* with a preface by Henry S. Pritchett, Ph.D., Sc.D., LL.D., Litt.D., President of the Carnegie Foundation for the Advancement of Teaching. 1926 (issued in 1927): pp. xxii + 692; 7¼ x 5 in.; one illustration; distributed free of charge on application, by letter or in person (the volume is "Bulletin No. Nineteen"). Carnegie Foundation for the Advancement of Teaching, 522 Fifth Avenue, New York City.

Physical chemistry and biophysics, for students of biology and medicine. By Matthew Steel, Ph.D., Professor of Biological Chemistry, Long Island College Hospital. 1928: pp. 372; 7¼ x 4½ in.; 38 illustrations; $4.00. John Wiley and Sons, Inc., New York City.

Fundamental principles of a systematic diagnosis of dental anomalies, with an explanation of a new classification based upon gnathostatic methods of investigation. By Paul W. Simon, Head of the Department of Orthodontics, Dental Institute, University of Berlin. *Translated* by B. E. Lischer, D.M.D., F.A.C.D., Non-resident Lecturer on Orthodontics, Dental School, University of Michigan. 1926: pp. 378; 6¾ x 4 in.; 189 illustrations; $5.00. Stratford Company, Boston, Mass.

The practice of periodontia. By Sidney Sorrin, D.D.S., Chief of Periodontia Clinic, and Samuel C. Miller, D.D.S., Instructor, Periodontia Clinic, College of Dentistry, New York University. 1928: pp. 160; 6 x 3½ in.; 122 illustrations; $3.50. MacMillan Company, New York.

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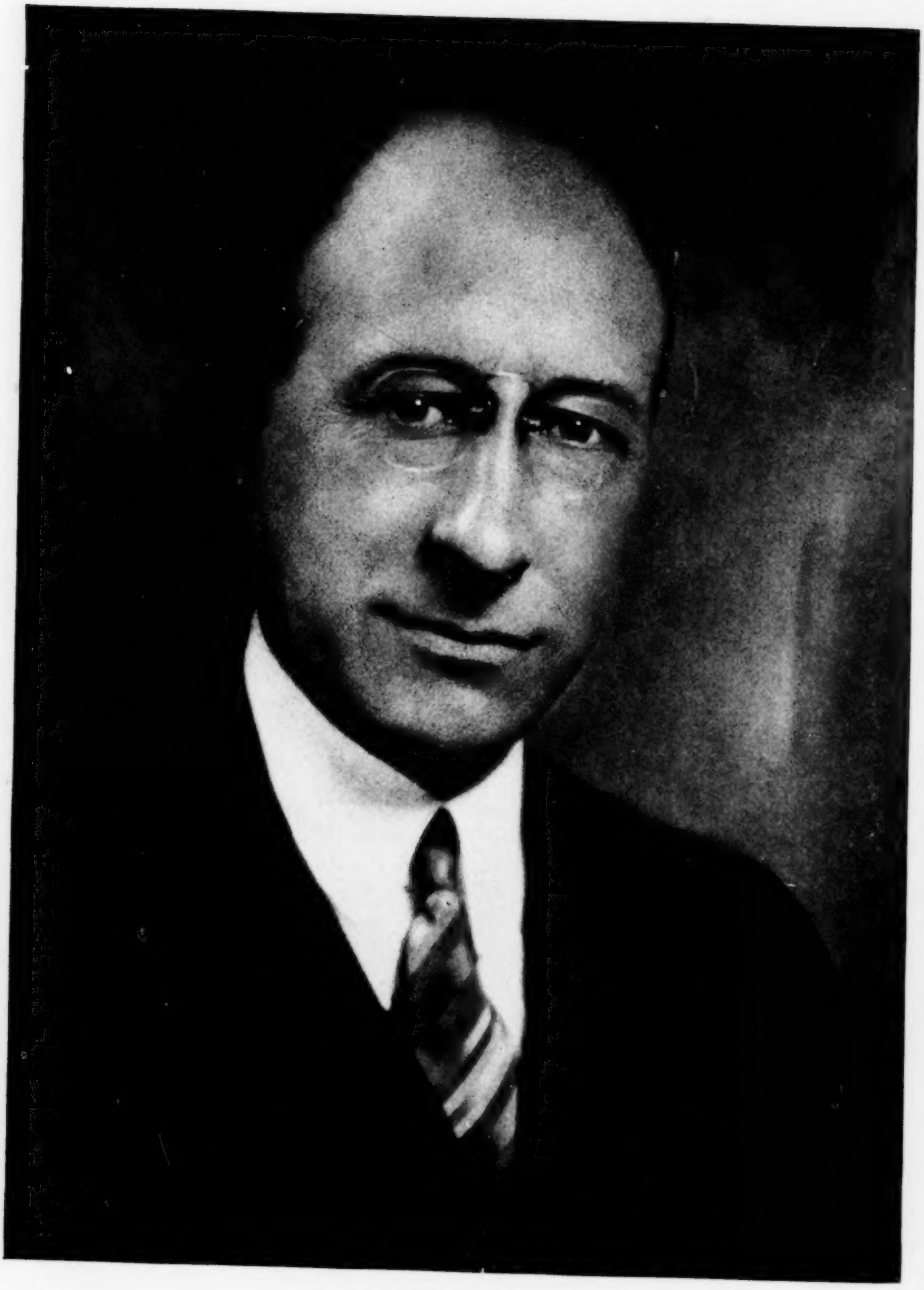

HOLMES CONDICT JACKSON

A TRIBUTE TO HOLMES CONDICT JACKSON

GEORGE B. WALLACE

New York University and Bellevue Hospital Medical College, New York City

The untimely death of Holmes Condict Jackson, Dean of the College of Dentistry, New York University, which occurred on October 25, 1927, has meant a real loss to dental education. Although in this field but a few years, he had already, through his character, executive ability, and scientific training, demonstrated qualities of leadership. Untrammeled by conventions and traditions, he brought a freshness and breadth of view to dental school problems and gave every promise of a career of great usefulness.

Dr. Jackson was born in New York City on February 18, 1875. His father, William Holmes Jackson, came from the north of Ireland. His mother, Jane Eliza Freeman, was of an old New Jersey family. On his mother's side was a direct inheritance from the Pilgrim settlers, he himself being of the ninth generation in direct line from John and Priscilla Alden.

He received his preliminary education in the Public Schools and the College of the City of New York, and in 1893 entered the Sheffield Scientific School of Yale University, graduating in 1896. At this time he had definitely decided on a career of teaching, and was made an instructor in biology at Yale. At the end of a year, finding his interests running strongly toward physiological chemistry, he transferred to this department. He did this largely because of his admiration for Professor Chittenden, the head of the department, and the leading figure in this country in physiological chemistry. He served three years under Professor Chittenden, during which time he obtained the degree of Ph.D. He spent the following year abroad working under Kronecker and Asher at Bern, and Hofmeister at Strassburg. He returned in 1901 and accepted the position of Instructor in Physiological Chemistry at the University and Bellevue Hospital Medical College, the medical division of New York University. In 1903 he

was made Assistant Professor and held this position until 1905. He then went to the Albany Medical College, and remained there until 1909, serving as Professor of Experimental Physiology and Physiological Chemistry. In 1909 he returned to New York University as Professor of Physiology. He retained this position until the time of his death.

Doctor Jackson had a wide interest in scientific subjects allied to medicine. He was the author of a Manual of Physiological Chemistry and of numerous scientific papers in his special field. He was a member of the American Association for the Advancement of Science, the American Society of Naturalists, the American Physiological Society, the American Society of Biological Chemists, the Society of Pathologists and Bacteriologists, the Society for Experimental Biology and Medicine, of which he was one of the founders and served as President for 1923–1925, the Harvey Society of New York, associate member of the New York Academy of Medicine, associate member of the First District Dental Society of the State of New York, and Fellow of the New York Academy of Dentistry. He was also a member of the scientific society, Sigma Xi, and was president in 1926 of the New York University chapter.

His civic interests were shown by his service as Food Administrator at East Orange during the war; his Presidency of the Men's Federation, East Orange, 1917–1919; and his membership in the Dental Advisory Board of the Department of Health, New York City. During his long residence at East Orange he became actively interested in the Munn Avenue Presbyterian Church, in which he filled the office of Elder. At Yale he was a member of the Theta Delta Chi fraternity, and at the Medical College of Nu Sigma Nu.

In 1901 he married Mary Abby Read, whose father was a well-known physician of Newark, and who, together with his mother and three daughters, survive him.

Dr. Jackson's interest in dentistry began when the project of making the New York College of Dentistry a division of New York University was proposed. He was made chairman of a committee from the University to study and report on the proposal. Dental education was a new subject to him, but he studied it with his characteristic energy and clear headedness. After a thorough survey

of the school itself, he passed to a consideration of dental education in general to determine whether the school could be brought to the degree of excellence demanded by the University. His study convinced him that the union would be mutually beneficial, and he accordingly recommended that it be brought about. On the basis of his report, in 1925, the Dental College was made an integral part of the University.

It was but natural that his clear exposition of the status of the school, of its problems and their practical solutions, should impress the University Council. In looking for one especially fitted to head the new division, the Council turned at once to Dr. Jackson and offered him the deanship. He accepted after some hesitation, for it meant giving up his very congenial work in physiology at the Medical School. His decision once made, however, he entered upon his new duties with much enthusiasm and gave to the work his entire attention.

He began with the conviction that dentistry, while retaining its status as an independent profession, should in fact be considered a branch of medicine. As a corollary to this he believed that the principles of dental education were the same as those of medical education. Thus he insisted on a sound preliminary education as an entrance requirement, and on the first two years of the dental course being given over largely to a thorough grounding in the basic medical sciences. The highly technical phase of the education would rest then on a firm foundation. He believed also that dental students, in order that they might have a broader view of the range of their profession, should have a usable knowledge of what is known as the "principles of medicine," and of systemic diseases into which oral pathology enters.

While these ideas were in no way unique, their successful carrying out is not usual. Jackson arranged with the University for a preliminary education that differed in no great degree from that offered to prospective medical students. In reorganizing the laboratory courses in the College, he drew largely on his own extensive experiences and made full use of the freely proferred services of his medical colleagues. Departments of anatomy, physiology, pathology, chemistry, bacteriology, and pharmacology were organized, each with a full time staff of trained teachers and investigators, and with ade-

quate laboratory space and equipment. The laboratory heads of the medical school became also heads of the dental laboratories, and the courses given were carefully outlined and supervised. He introduced also courses which served to further the general professional training of the students; thus lectures on the principles of medicine and on diseases of the nose and throat were given by experienced teachers from the Medical College.

In the more specialized side of the curriculum he made no radical changes, for this field was too new to him to allow of fixed opinion. In order to inform himself he visited many of the other dental colleges and carefully studied their methods of instruction. He freely consulted dental authorities whose opinions he valued, in particular his old time friend and Yale associate, Dr. Gies, who had completed his exhaustive survey of American dental colleges, and who was of the greatest help to him. As his knowledge began to take form, he very deliberately started to introduce improvements and innovations into the clinical curriculum. He rearranged the existing courses, curtailing some, enlarging others, and bringing them all into a more harmonious relationship. He kept in mind constantly the plan of a well rounded education, in which no subject should stand by itself but each should be a correlated part of the whole.

He realized at the outstart that while system and organization are essential for the success of any school, it is the personality and calibre of those who do the teaching that give the school its particular degree of excellence; that the student's desire for knowledge, his ambition to be master of his subject, and his ideals of professional behavior are dependent largely on the stimulus and example offered by his teachers. Although he started his work with an admirable faculty, he began to enlarge it, always insisting that only those be added who were outstanding figures in dentistry. With such as heads of departments, the success of the school would be assured.

Dr. Jackson believed that the dental school should have a broader sphere than educating students. It should also perform a definite public service. Following this idea, there was established a Saturday clinic for children; the College entered into relations with Bellevue Hospital, the great city hospital of Manhattan; it took part in the Yorkville-Bellevue Health Demonstration program. He had hopes

also of making the College of greater service to the community living or working in its vicinity. In addition to this broader outline there were set aside alumni days on which advances in the practice of dentistry were shown by means of demonstrations and lectures. Plans were also prepared for an extensive and thorough postgraduate course.

It may be assumed that any faculty whose constituted head has a character which enforces both liking and respect will unreservedly work with him toward a desired end. Dr. Jackson had the loyal support of his faculty, and he maintained this because he gave to its members his loyal support in return. Loyalty is a much abused term, but in this case it meant the whole-hearted support given to a man who was not actuated by an idea of personal advancement, but was earnestly giving his all to a common purpose.

Only his intimate friends knew that during the last year and a half of his life Dr. Jackson was suffering from a malady which could have but one end. His physical condition during most of the time was one of great discomfort, and he was forced to give up a social side of life which had meant much to him. He was under constant medical observation and treatment, and on several occasions was obliged to spend days in a hospital. He accepted his fate without complaining, and continued his work with unabated courage and determination. A true estimate of his character is obtained through the light afforded by such behavior.

SYMPOSIUM ON PREVENTIVE DENTISTRY[1]

FRANK A. DELABARRE, A.B., D.D.S., M.D., Boston, Mass.; HOWARD R. RAPER, D.D.S., F.A.C.D., Albuquerque, New Mexico; C. J. HOLLISTER, D.D.S., Harrisburg, Pa.; and ARTHUR H. MERRITT, D.D.S., F.A.C.D., F.A.A.P., New York City

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I. PEDODONTIA. FRANK A. DELABARRE, A.B., D.D.S., M.D., BOSTON, MASSACHUSETTS

The American Association of Dental Schools, at the last meeting in Chicago, vainly spent some time trying to define preventive dentistry. It was even stated that all dental procedure is preventive; in one sense it may be, but nevertheless its work is curative and reparative rather than preventive. The distinction necessitates a definition of the respective fields and it is axiomatic to say that *prevention is effective in inverse ratio to the age of the patient.* It follows, then, that preventive dentistry must be practised on children. An age limit must be established. It is not practical to practice either to the exclusion of the other. It is true that dentistry for children gives the maximum of prevention with the minimum of operative procedure. Then, the logical dividing line is the period of adolescence, and, stated in terms of physiologic age, the eruption and functional placing of the second permanent molars should mark the termination of the preventive phase of dental work.

There are specialists in children's dentistry, and there is great need for more, but every general dentist who is qualified by temperament to handle children, with the understanding and sympathy demanded by their age, interests, and mental outlook, should give this service.

[1] Read at a meeting of the First District Dental Society of the State of New York, New York Academy of Medicine, February 7, 1927.

It is time to stop neglecting the child. Emphasis is placed on the cardinal requirements for the successful practice of pedodontia:

1. Enroll the child as a patient by the age of three.
2. Appreciation of the psychological approach to the child.
3. Periodic and systematic series of appointments during the period of growth and development.
4. Proper division of responsibility between the operator and the home.
5. Rendering a complete service from the standpoint of *health*, general as well as local, educational and advisory as well as operative.
6. Careful attention to developmental dental defects and initial lesions.

It is desirable at this time to decide upon a name for this work in order to avoid confusion and the adoption of a misnomer, such as "dentistry." In the early days of our profession that name was applicable and fitting, but in the light of present knowledge and practice it is too limiting. Because of the inherent dependence of dentistry upon medicine for the interpretation and care of the vital processes of life, any new conception of our responsibilities in the maintenance of health should be closely related in definition and application to the usage of the larger profession, of which we are, at most, a limited specialty. Preventive medicine and preventive dentistry should be parallel in name as well as effort. Preventive medicine is sanitary science, and its application is mainly to the public in the mass, and incidentally to the individual.

Preventive dentistry, today, must be applied to the individual, because of our limited knowledge of the cause and control of dental disease. When we know more of such causes, which have undoubtedly crept in with civilization and are closely related to diet, metabolism, and manner of living, or environment, we may be able to deal with the mass. Medicine already has a specialty which closely parallels the field we are considering, which aims, as we do, at prevention, and which is applied to the individual, namely, pediatrics. Is it not more logical then, to conform to medical usage and practice, and call it "oral pediatrics," or "children's dentistry," or "pedodontia?"

The orthodontists unanimously and enthusiastically welcome the advent of prevention. Resolutions were adopted at their 1925

convention, at Atlanta, in which it was affirmed that: "It is believed that the best possible future development of dentistry must be along the applied principles of prevention." And further: "The American Society of Orthodontists makes this unanimous appeal to the dental profession at large to use all their influence to bring about, as speedily as possible, the consummation of such development through individual effort, and the adoption of a revision and balancing of dental curricula that will emphasize the teaching and practice of pedodontia." The official vote thus recorded voices the opinion of every orthodontist, individually, because the main endeavor of that specialty is the restoration of function as a fundamental step in the securing and maintenance of local and general health.

Orthodontists are quite generally attempting to apply the same principles to their own work, following the teaching of the late Dr. E. A. Bogue, of New York City, in securing for the child of five, who presents evidence of arrested development, a more adequate growth. Malocclusion of the deciduous or mixed dentition almost always develops into an adult malocclusion of a progressively complicated type. Common sense demands preventive orthodontia as soon as a positive diagnosis is made. Functional efficiency depends, in large measure, upon a full complement of healthy teeth. Malocclusion is usually a result of several causes, some of which are basic disturbances of vital organs or functions, resulting in like maldevelopment of the bodily structure, while others are local in origin and effect. By far the most common and important of these local causes is neglect of the teeth in early childhood.

No orthodontist can start his work without having the mouth, if crippled, put in a healthy condition first by the dentist. This results in serious delay, sometimes at a critical period of development. Also prolonged dental attention during orthodontic work is a serious interruption in the continuity of the work. Premature loss of any of the deciduous teeth deprives the orthodontist of the opportunity to apply stimulus for growth in that particular region, and complicates the technical problem. Loss of a permanent tooth is a calamity that renders impossible the attainment of full functional efficiency and compels a compromise treatment. Where the condition of loss of teeth has been neglected, and no space retainers applied, the usual

result is a migration of the remaining teeth, producing asymmetrical relation tremendously complicating the case and adding technical difficulties that demand the utmost skill and also a disproportionate amount of time in correction.

Above all in importance is the bad general effect that follows the neglect of a child's mouth. The lowered physical tone and development of the body due to poor mastication and consequent malnutrition are big handicaps for the orthodontist to overcome. It must be remembered that the child needs good food and function, not only to sustain life and provide for normal tissue repair, but also to afford added material for intensive growth and development. Mental retardation is another effect of early dental neglect, generally recognized and accepted, and brings one or more important items for the consideration of the orthodontist.

To sum it all up: the orthodontist needs the skillful help of the pedodontist before, during, and after his treatment, if an efficient occlusion is to be obtained. But children's dentistry does not need the approval and support of our specialty to prove its worth. No other branch of our profession can show such startling results. The wonder is that it has been so long in coming. Eighty per cent of caries can be eliminated, and extraction reduced to zero, as has been proved by the experience of the Forsyth Infirmary.

Many individuals have contributed to the furtherance of this idea, but it needed Hyatt to call our attention to non-carious pits and fissures, and Bödecker to furnish the scientific evidence of the necessity for prompt operative procedure to *prevent* caries in these most vulnerable areas.

II. RADIODONTIA. HOWARD R. RAPER, D.D.S., F.A.C.D., ALBUQUERQUE, NEW MEXICO

Until rather recently we have not thought of radiodontia as playing any part in preventive dentistry. It has, in fact, played no part, further than to reveal, in a striking manner, the urgent necessity of prevention. X-ray examinations, as my friend Simpson aptly puts it, have been in the nature of autopsies. But I doubt if it is putting it too strongly to say that the clinical preventive dentistry of tomorrow will be based on x-ray examination. Not the ordinary periapical

examination, which reveals the evidence of dental disease when it has already had time to exert an untoward systemic effect, but the *interproximal x-ray examination*, which focuses attention on the region where the dental disease begins.

The aim of the future clinical preventive dentistry must be, I believe, the prevention of toothache, i.e., the prevention of pulpitis, pulpless teeth, and the systemic disease caused by such teeth. Toothache is preventable, but dental caries is not—not, at least in the same sense or to the same degree as toothache. It is ironic, and yet human, that so much should be said about preventing caries and so little about preventing toothache.

Those who would prevent dental caries may be classified into two groups: the dietitians and the advocates of various methods of cleaning. Inquiry into what the dietitians have to offer leaves much to be desired. We learn that to prevent caries by diet one should start with the diet of the prospective mother, or grandmother. A good suggestion for posterity. But it is rather impractical, not to say discouraging, for a man who wants to prevent decay of his own teeth, to be told he may hope to accomplish it by regulating his mother's diet before his birth. As for a method of cleaning teeth which will entirely prevent all decay in anything like a large proportion of mouths—there is no such method. Cleanliness will lessen the amount of caries, we hope and believe, but the average person of today cannot reasonably hope to escape all decay of his teeth.

Briefly then, the best dentistry can do, by way of preventing caries, for people already born and in possession of their teeth, is to say: "Eat a good mixed diet and keep your mouth clean. This will tend to lessen decay, but, if you are an average person, it will not prevent it altogether. So watch out." Toothache, on the other hand, can easily be prevented. Ninety per cent or more of people can escape it altogether. Not ten per cent can escape all caries.

Much has been written in recent years about diseased teeth causing disease of the body. The subject is usually discussed in terms of infection. Instead of discussing it in the usual manner, suppose we consider it here in terms of tissue destruction—which is the manner in which a radiodontist sees it.

Perhaps the most common characteristic of all disease is tissue

destruction. Pulmonary tuberculosis destroys the lungs; gastric ulcer attacks the lining of the stomach; cancer attacks almost any tissue of the body; wherever there is suppuration and, to a lesser degree, wherever there is inflammation, there is tissue destruction; nephritis attacks the kidney; syphilis attacks the central nervous system, the bone; and so on. The loss of body weight so common to all diseases is but another manifestation of tissue death and destruction. Viewing disease, then, as a tissue-destroying force, dental disease has a portentous beginning, starting as it does with *caries* of the enamel; that is to say, with dissolution of the hardest substance in the body; and then, as it progresses, attacking the next hardest substance in the body, the dentin. If unchecked, it soon reaches and destroys the pulp—thence out through the root of the tooth into the bone, destroying it. It is the present belief that from the focus in the bone the disease may now spread to almost any tissue or vital organ of the body, manifesting its presence always, at bottom, in the same way, that is, by tissue damage and destruction, causing nephritis, gastric ulcer, endocarditis, and other serious diseases. Such is the progress of disease, entering the body by means of the dental path, as viewed by the radiodontist. The dento-alveolar abscess is like cancer of the skin in that the consequent danger lies not in the lesion itself, but in the development of metastatic, tissue-destroying lesions elsewhere in the vital organs of the body.

When a man dies of disease, he does not die all over at once. He is not all alive one minute and all dead the next. The process is progressive. He dies in parts, so to speak. And when enough parts are dead, the organism as a whole dies. Shortly before death practically all organs have started to disintegrate—as the common phrase has it: "there is not a sound organ left in the body." All disease, then, is the beginning of death. True, final death of the organism may be a very long way off, but any pathological destruction of vital tissue is a beginning, and, on principle, should be stopped as soon and as definitely as possible.

With the idea, then, of keeping disease out of the vital organs of the body, let us consider the matter of treating dental disease. These facts are immediately obvious: we can stop the progress of dental disease, with mathematical certainty, *before* toothache, i.e., before

pulpitis; and we cannot stop it with absolute certainty *after* toothache. The common ailment of toothache, then, is seen to occupy a most significant place in the disease process outlined in this paper. Its pathologic significance lies in the fact that it marks the point where disease, entering the body by the dental path, first reaches the circulation, first gets *inside* the body. Its strategic and clinical significance lies in the fact that before toothache the prognosis for effective treatment is most excellent; after toothache, the prognosis is never better than doubtful and, as usually handled by the profession, is bad.

Obviously, the chief aim of both patient and dentist in the care of teeth should be to fill, and keep filled, the carious cavities in teeth before they become too large. By so doing dental disease can be kept from reaching the pulp; and so toothache is prevented, and disease is kept not only out of the pulp, but also out of the bone and vital organs —"prevent the toothache and you save all," to adapt the paint manufacturer's slogan. Cavities in teeth must, of course, be found before they can be filled. And so we come specifically to the part the x-rays are to play in practical clinical preventive dentistry.

The question immediately arises: are the ordinary methods now in common use such as to enable dentists to find all cavities in teeth? That question may be answered in the affirmative so far as the open, exposed surfaces are concerned. But how about the hidden surfaces, the surfaces between the teeth, i.e., the approximal surfaces? All dental radiographers of any experience or discernment know that a large proportion of the cavities in the proximal surfaces in teeth escape detection by the ordinary ocular, instrumental methods of examination in common use. Radiographers know this because proximal cavities that have escaped detection by ordinary examination are revealed by x-ray examination.

The relative efficacy of the x-ray examination as a means of locating proximal caries, compared to the ordinary methods, was put to a test by this writer, with the following results: A young lady of about twenty, with rather good looking teeth, was selected as a patient. An x-ray examination of her mouth and teeth revealed the following findings: one large cavity, two medium size cavities, two very small cavities, one filling with caries going on underneath it, one filling improperly placed with an excess of filling material encroaching on

the interproximal tissues—in all, seven findings. After the x-ray examination, the patient was examined by ten dentists by ordinary ocular and instrumental methods, with the following results: two dentists noted two of the seven findings; six, only one of the seven findings; and two, none of the seven findings—a one hundred per cent failure on the part of ten representative dentists to find all, or even a high or moderate proportion, of the proximal lesions revealed by the x-rays!

While I consider the foregoing experiment significant and indicative of the existing facts, I do not place too much emphasis on it, or ask others to do so. Such tests are so likely to prove what the tester wants to prove. The fact—for I know it is a fact—that dentists do not find proximal cavities until they frequently endanger or expose the pulp is perfectly apparent to anyone who will simply look at a skull, or a photograph of the dentures of a skull. Even with the soft parts gone, it is impossible to see between the teeth, from the labial or buccal, with any degree of satisfaction; and it is still less possible to see between the teeth from the occlusal. Yet it is the common practice to look from the occlusal, where it is exceedingly difficult to see the proximal surfaces. Is it not perfectly obvious that the practice is not to look *between* the teeth at all, but to look *at* the occlusal surfaces; which is to say, we wait until the cavity has extensively undermined the occlusal enamel. I recently asked a dental colleague to examine a patient's teeth by ordinary ocular-instrumental methods and be sure to tell me if there were any proximal cavities. He knew the teeth were to be radiographed later. He found three "suspicious" places. Under radiographic examination, one of the suspicious places was found to be a cavity; the other two were not. In the absence of the radiographic examination, would my friend have separated the teeth and examined more carefully all three of the suspicious places? He probably would not. He would have waited, if he is like most practitioners, for the occlusal enamel to "look worse," or break down; or (mal-practice) for the teeth to ache.

But I labor to make my point. It is to be expected that any new method will be held necessary by some. It is almost unbelievable now, but it was only a few years ago that many dentists said they did not need the x-rays for dental diagnosis at all, and that many physi-

cians said they did not need radiographs of the chest—that they could find out all they wanted to know with a stethoscope. The wise physician of today uses *both* the radiograph and the stethoscope, just as the wise dentist will, I believe, use *both* radiographs and ordinary methods for examination of the interproximal regions.

If it is true, as I contend, that dentists fail to find proximal cavities, and that these cavities can be found by the use of x-rays, then why are not the x-rays more generally used for this purpose? There are two main reasons: (1) only the dentists who have done a considerable amount of x-ray work realize the extent of the failure to discover proximal cavities by the ordinary instrumental and ocular methods; others are laboring under the delusion that they are finding these cavities, just as they labored under the delusion that they were treating pulpless teeth correctly before the advent of the x-rays in the practice of dentistry. (2) Until recently it has required ten to fourteen or more exposures to make an x-ray examination of the teeth, which is but one way of saying that such examinations were too difficult and too expensive to be done periodically for people of moderate means.

The latter difficulty has recently been overcome by the development of a new type of x-ray examination, which I have called the interproximal examination and which can be made in one-half the number of exposures heretofore required, with a corresponding reduction in difficulty and expense. The new examination is based on the simple and obvious idea of radiographing both the upper and lower teeth (the coronal two-thirds) simultaneously. It was announced over a year ago and is now commencing to be used rather extensively. Because it is a simplified and therefore less expensive examination, it can be made periodically at intervals of from one to two years. If the teeth are kept under such observation, there is almost no danger at all of any cavity becoming dangerously large before detection. Aside from its value as a means of finding cavities, the periodic interproximal examination has, to my mind, the very great added advantage of being something tangible to which the idea of preventing toothache may be tied.

Perhaps I should mention that this new interproximal examination is in no sense calculated to take the place of the older familiar periapical

dental x-ray examination. It is made for a different purpose. It is made to prevent teeth from becoming pulpless. The periapical x-ray examination is made mainly to observe such conditions after they have been allowed to occur. If a patient needs a periapical examination, this new examination cannot be substituted. On the other hand, a very great number of people need the new examination who do not need the old. Indeed, every person with teeth, who wants to keep them at the minimum expense and the minimum risk to health, needs the new examination made periodically.

Time will not permit me to dwell upon it, and I do not care to anyhow, but I should not fail to mention that the interproximal x-ray examination reveals incipient periodontal disease; and, to my mind, the hope for permanent curative benefit in the treatment of this disease depends upon early diagnosis. But it is the pulpless tooth problem I am discussing, not periodontal disease.

Once pulpless teeth are allowed to develop, the patient can acquire as many different opinions about them as he has money and patience to patronize different dentists and physicians. The disagreement among dental and medical practitioners regarding pulpless teeth is very great, and, to the best of my ability to judge at this time, permanent. Observing this disagreement, patients are losing faith in the opinion of anybody on the subject, and are taking the matter more and more in their own hands, refusing to have teeth extracted until serious illness overtakes them. Unfortunately the radiograph does not afford a basis for agreement as to what to do with pulpless teeth, and, I fear, never will. But it does offer an adequate basis for the prevention of toothache and so the prevention of the occurrence of the pulpless tooth.

I sometimes wonder if the prevention of toothache can actually have the importance it seems to have in the light of our present knowledge regarding degenerative disease; if the mere filling of a tooth can actually prevent, say, a heart lesion. And, frankly, I am inclined to doubt it. It seems too good, too simple, too easy to be true. Then I think of the practice of dropping a silver salt solution into the eyes of a new-born baby. What could be simpler? And yet this simple act prevents blindness. The filling of a tooth is most intricate by comparison. The average person looks upon toothache as something

inescapable, like death and taxes. We of the dental profession know that this is not true, and it is our obligation to teach the layman. Also we should teach him that toothache means something more than just a severe pain. Once it is allowed to occur, it means either the loss of a tooth or the retention, at considerable bother and expense, of a tooth which has now become the kind of a tooth which may be a source of systemic disease.

Papers on the subject of prevention frequently close with phrases like the following: "Our work is, of course, inconclusive. We have, in a sense, just started, but we have progressed far enough to cause us to hope"—and so on. Such statements are, of course, both necessary and honest; but, stripped of their polite formality, they would read simply: "We are sorry we have nothing at all of value to offer the clinical practitioner." I am exceedingly fortunate that I do not have to close my paper that way. There is nothing inconclusive or intangible about the interproximal x-ray examination, or the prevention of toothache it stands for. The question is not "have we" or "can we," but "will we," do that which so obviously lies within our reach.

III. SCHOOL DENTISTRY. C. J. HOLLISTER, D.D.S., HARRISBURG, PA.

Scope of the corrective dental clinic

The first dental clinic service furnished in a school that we know of was provided through Ebersole's activity in the Marion School, in Cleveland about 1912. I feel sure that most of you are familiar with the dramatic results attained by that experiment, so I will simply state that it indicated very definitely that the condition of children's teeth has much to do with their mental and moral development. When we note the thoroughness with which that work was accomplished, we are forced to admit that such a program would be impossible on the great mass of school children. That experiment reached only twenty-seven children. The corrective clinics that have been established since that time, all over the country, have been in the main emergency in the type of service rendered, and conducted virtually on a charity basis. Leading educators as well as professional investigators have frequently stated that charity corrective dental service has no place in the public school system, and that the emergency service offers

only temporary relief, doing little or nothing to prevent the need for such service. The corrective dental clinics, as I observe them, are serving just about as outlined above. The majority of them are simply taking the worst cases upon recommendation of school nurse or medical inspection personnel, and doing extractions and other forms of relief work.

Plans for operation and financing

There are numbers of definite methods for financing and operating school dental clinics. The vast majority of these school dental clinics have been started by funds contributed through charity and service organizations, and are manned by local dentists volunteering their time. Where school dental clinics have been started in this manner, quite frequently, after a year or more of operation, school boards have taken over such clinics, hiring a dentist either on an hourly basis, half time or full time. The school dental clinic, even with its inadequate service, has been of great benefit, doing a vast amount of good to the individual children receiving service, and has resulted in a great awakening to the need of the broader and more effective type of school dental service which I will later describe. In some communities under my jurisdiction, the following rule has been adopted where a full-time dentist is employed by a school board. By coöperation with deans of dental colleges a good man is selected from the senior class, and the following September placed in charge of the clinic at a salary of $1,500 or more a year. If his work has proven satisfactory in the judgment of an advisory committee to the school board, composed of local dentists appointed by the local dental society, he is retained for a second year at an increase in salary of from $300 to $500. At the end of the second year, however, regardless of how satisfactory his services have been, he is automatically released and the procedure repeated. The reason for such action is that the clinic is supplied by a well recommended ambitious young dentist with ability, who serves with enthusiasm the first year and continues his enthusiasm the second year due to higher compensation among other things. But if he be a man with ambition and vision, he could not afford to continue longer than two years at the compensation which it is possible to pay in school clinic work. Hence, if he stays more than two years, he very likely would be a type of man not wanted in such a position.

Now let me give one illustration as to the adequacy or inadequacy of the corrective dental clinic in schools. In the city of Philadelphia we have ten or eleven dental clinics operating full time for the benefit of school children. There are something over 250,000 school children in that city, and if the generally accepted percentage of children needing service holds in Philadelphia, at least 90 per cent of them need corrective dental service. It is readily seen, then, that ten full-time dental clinics would little more than scratch the surface. In other words, the dental clinic service in Philadelphia has proven itself to be an emergency proposition. For instance Johnny, with an aching tooth, can go in to a dental clinic; he is suffering and incapacitated. He goes in and receives service. His little play-mate sitting beside him may have and probably does have need of corrective service, but he can not go in. He must wait until his tooth is broken down almost, if not entirely, beyond repair before he can go in. Such service is a cart-before-the-horse procedure—locking the door after the horse is stolen. Those in position to know make this assertion, that while the corrective dental clinic has been of great service, it is entirely inadequate to meet the problem at hand.

Education

Education is the fundamental principle of prevention in dentistry. I picked up a railway journal some time ago, and found there an editorial by one of our leading railway executives in which he said: "If we are to lower the frightful toll of human life taken at railway crossings the only way that will be accomplished is by the education of the motoring public." That is a truism which applies to matters of health as well. Some one has very aptly said: "To cure is the voice of the past, to prevent is the divine whisper of the future." The statements that I now propose to make are not simply a matter of personal opinion hastily formed, but are the product of a number of years of close study and observation.

The State Department of Health of Pennsylvania has for quite a number of years been doing everything in its power to induce local school boards to employ dental hygienists to do practical prophylaxis, and teach the children fundamentally what to eat and how to eat it, and to stimulate interest in the proper use of the toothbrush. May I

here give you an outline of what a dental hygienist is. She, as you know, is a young woman who is trained to clean teeth; she does not extract nor does she fill, she does not even make a definite diagnosis of conditions found in the mouth, she simply cleans the teeth. But she is far more than that; she is the teacher of the "how, why and when of the toothbrush," also fundamentally of nutrition. The biggest phase of her work—and this particular phase I have seen exemplified almost 100 per cent—is the molding of the plastic mind of childhood, training children in habits of cleanliness, both dental and general, that will stand them in good stead the rest of their lives. The Bible tells us: "Train a child in the way that you would have him go, and when he is old he will not depart therefrom." Or, quoting a high church dignitary in Philadelphia a few years ago: "Give us the child until he is seven years old, and you may take him where you will, we care not, we know where his heart will be." We all know the truth of the above quotations. I have proof of them in my own home.

I have four daughters ranging in years from nine to two. From the time the first one was two years old, their training by their parents has been such that now not one of the four would forget to clean her teeth after each meal. In addition to this training their parents have taken each of them to a dental office twice or three times a year; also they have planned and supervised the diet of these children, so that none of them have ever experienced a real toothache and it is not likely that they ever will. Now, if the above described care and treatment will insure for my children good teeth and by that means good health—if such training and treatment can be given to all children economically and efficiently, I believe we owe it to them. I could recite numerous examples proving that the dental hygienist is the medium by which such training can be given to large groups of children efficiently and economically. We find that the costs are little more than fifty cents per year per child to provide them with dental hygiene service, Boys and girls behave better when they have clean teeth. I once made this statement before a group of dentists. In a discussion which followed one of the dentists is quoted as having said: "I feel that I have learned something worth while tonight. I have a boy at home that I have punished in every conceivable way, still I cannot make him behave. I am going home now and clean his teeth." While

that dentist has not reported to me the result of the boy's treatment, I feel safe in saying that that boy is a better behaved child because of that treatment. The following experience which came to us in our demonstration work will prove the point.

I had taken a dental hygienist with her portable equipment to the town of Scottdale, Pennsylvania, for a demonstration to the schools, the school board, and the public. The first child presented for a prophylactic treatment by the dental hygienist was one of the dirtiest urchins I have ever seen in a school building. As he approached the dental hygienist's chair, he was as fresh, surly, and unwilling as he was dirty. He said with almost a snarl: "I have been sent up here to get my teeth cleaned." The dental hygienist looked at him, and then with womanly tact said: "I cannot work for you, you are too dirty," but she said she did not pass him up. She took him by the ear and led him to a convenient wash stand. After talking to him a little while, he tried with the soap and nail brush to remove the real estate from beneath his finger nails. He shampooed his hair, washed behind his ears, all unusual procedures I assure you; then, smoothing his hair down the best he could, he presented himself at the chair like a little gentleman to receive the service she had to render. She cleaned his teeth, taking about a half hour to do the job and talking to him all the while, telling him not only to take better care of his teeth but to take better care of his whole body, to try to wear cleaner clothes, to study a little harder so that he might be at the head of his class instead of being two years back of the grade he should have been in according to his age. In the meantime his teacher had come into the room to observe what was going on. After this boy was dismissed from the chair, without being prompted by anyone, he turned to the dental hygienist and said: "Thank you." His teacher turned to me and said: "That boy has been in my room for nine months, and that is the first sign of courtesy I have ever seen him show."

The story might end there, but I spoke before the Scottdale Rotary Club that noon telling the story of this boy as far as I have related it here. After the meeting of the club they voted to go in a body to the school house, to visualize the things I had been talking about. As 25 or 30 of us approached the school ground, this boy spying me ran to me with hands outstretched to show me how clean they were; then, probably

having been prompted by someone, he came to boy scout attention, waiting for me to comment on what he knew I saw. I looked him over; he had all clean clothes from head to toe; was wearing a necktie, something that I had been told he had never been known to wear before; his hair was plastered down like that of a sheik in the movies, and his face fairly shone. He proudly went into his blouse, and pulled out a new toothbrush and tube of paste. I learned later that one of the Rotarians had slipped into a store on the way up and supplied him with those articles. The story again might end here, but I have relatives in Scottdale, and in a short space of about two years I learned that that boy had made up the two years he was behind in school, and now stands well toward the head of his class where he belongs. Without any attempt at dramatics I want to say to you that the dental hygienist and myself get a real thrill from the knowledge that our contact with that particular boy has meant the salvaging of his life to better things. Your imagination does not have to travel far to picture what might have happened to him had he continued as he was before we saw him. He probably would have drifted down and down, eventually as he became older getting into trouble and becoming a guest of the State behind iron bars. As it is now, he is on his way to decent American citizenship, all because if you please, he had his teeth cleaned. This may seem to you a dramatic case, but I have observed it exemplified with varying degrees in practically every contact the dental hygienist makes.

One of the most important parts of the equipment of a dental hygienist is a hand mirror. When a child is placed on the chair, he is shown how badly he needs attention. Some twenty or thirty minutes afterwards he is again allowed to see his teeth. He steps from the chair with his chest and head held higher, his eyes brighter. He has a different viewpoint on life entirely. In other words, his self-interest has been awakened. You and I did not begin to wash back of our ears until we began to go to see the girls. School prophylaxis awakens your self-interest a great deal earlier than would otherwise ordinarily be the case. I make the following statement without fear of contradiction, that the dental hygienist has proven herself to be the biggest and most effective thing that has come into school health work in a long, long time, in that she is an immediate visualization of a benefit

derived. You cannot put over any program for group or individual betterment without the enthusiastic interest of the individual and group.

I do not think, in my enthusiastic endorsement of the dental hygienist, that she is the panacea for all human ills. There is something far more fundamental than merely cleaning the teeth, and that is what we eat and how we eat it. I do not presume to try to enlarge on this great fact. Howe, McCollom, and others have scientifically proven the effect of diet on the teeth of animals. I am reminded here of a story apropos of the subject. A woman on the advice of her physician went on an extended fast. During the first ten days of this fast she received little or no newspaper publicity, but after the twentieth day notice of her activities had front page space in the newspaper, on the thirtieth day she was in heavy type on the front page, on the thirty-third day she received a large bundle of letters containing proposals of marriage—all of these letters were postmarked Scotland. Seriously, if we ate what we should, the way we should, the vast majority of human ills would be unknown.

Observing wild life in the animal kingdom, we are able to note that animals living in their natural environment do not die of disease. They die either a death of violence or of old age. When they pass by the old age route, it is not the heart, lungs, or some other vital organ which gives out first, they simply die all over, like the "one hoss shay" of poetic fame. The human being does not go that way, and almost invariably the teeth go first. None of you have seen many adult human beings with perfect dentition. So seldom do we find such a conditon in adults that, when these cases are discovered, they are usually given front-page publicity. It is my firm belief that, if the nutrition of the expectant mother from the time of conception until birth could be supervised and her diet also controlled during the nursing period, and if the child's diet were under strict supervision until he attained the age of twelve, he would be assured of an almost perfect set of teeth throughout his life. Dr. Howe here would say that that is true with reservations. His reservation would be that a normal proper dietary must be constantly observed. To meet his statement, may I say that a child twelve years of age, who had been all his life under such a supervised diet, by force of habit logically

would continue. Hence, I feel sure that I am not too idealistic in my claim.

Conclusions

The Department of Health under which I serve was convinced of the logic of the plan pursued by Dr. A. C. Fones, of Bridgeport, Conn., and that is the reason for the existence of the Division I represent in that Department. Most of you are personally acquainted with Dr. Fones and are familiar with the findings made public in his pamphlet called "The Bridgeport Five Year Report." Some of you have, no doubt, investigated and have verified claims of results made by Dr. Fones in Bridgeport, and I am sure that the profession throughout the country accepts his claims as reasonable. Such being the case I want to say, with all the force at my command and backed up by the actual facts, that in Pennsylvania we have over one hundred and thirty-five communities which in varying degree are absolute confirmation of the results of the Bridgeport plan. In other words, we have over one hundred and thirty-five little Bridgeports in Pennsylvania. Those that have had this service four years or more would be able to give percentage results uncannily close to those presented by Dr. Fones.

The profession of this city, state, and nation have a big obligation to the children of our country. And until some better means has been devised and tried, dentists should bend every effort to secure the employment of at least one dental hygienist to every one thousand school children, to attack the problem of the frightful toll of economic loss occasioned by defective teeth among children. My plea is for the hope of our nation, the children. I may properly close my remarks to you with a little verse which, to my mind, exemplifies the obligation of the dental profession to humanity:

I took a piece of plastic clay,
And idly fashioned it one day.
By my fingers' skill,
It bent and yielded to my will.

I come again when days are past,
The bit of clay was hard at last.

My early impress still it bore,
And I could change its form no more.

You took a piece of living clay,
And gently formed it day by day,
And molded, with your power and art,
A boy's soft and yielding heart.

You come again when years are gone,
It is a man you look upon.
Your early impress still it bore,
And you can change him never more.

IV. PERIODONTIA. ARTHUR H. MERRITT, D.D.S., F.A.C.D., F.A.A.P., NEW YORK CITY

Periodontia is that branch of dentistry which concerns itself with the study, prevention, and treatment of diseases affecting the supporting tissues of the teeth. Inasmuch as it deals with that which is most fundamental in dentistry, it is only logical that any inquiry into the present day possibilities of preventive dentistry should begin with periodontia, for unless it is successful in maintaining health in the tissues which surround and support the teeth, all else in dentistry is like a home that is built upon sand; it cannot endure.

Success in the prevention of any disease depends in large measure upon a knowledge of the facts that enter into its etiology, and upon their removal. Without this knowledge, and until such factors have been removed, only partial success can be expected. The etiologic factors in disease may be arbitrarily divided into two groups: those that predispose to disease, of which heredity, nutritional abnormalities, certain diatheses, etc., are examples; and secondly, local exciting causes. In the first group are to be found those subtle and illusive factors that precede and prepare the way for disease without the concurrent influence of local exciting factors. Both play their part in diseases affecting the periodontium. Malformed jaws, irregular teeth, frail alveolar support, etc., all predispose to disease, and are representative of man's heritage from the past. Likewise, disturbance in nutrition may be a predisposing factor in periodontal disease, in that, among other things, it lowers resistance to infection, and, in

so doing, increases the possibilities of disease through the activity of the local exciting factors. Both heredity and abnormalities in nutrition are representative of some of the unsolved biological problems underlying diseases of the periodontium, and which militate against, and make more difficult, any program for preventive dentistry. They are not, however, the controlling factor.

Fortunately, knowledge regarding the local exciting causes is more complete and more definite. In a general way these are known and, as a rule, are not difficult to control, provided their etiologic relationship is understood. Chief among these, and underlying all periodontal disease, is low resistance to infection on the part of the cells composing the tissues of the periodontium, brought about in large measure through failure actively to use the teeth and jaws in mastication, plus the possible influence of some of the predisposing factors already mentioned; and secondly, the invasion of these tissues by the bacteria of the mouth, principally the streptococcus and the staphylococcus. So important are these two factors (low resistance and bacterial invasion) in the etiology of periodontal lesions that it is not too much to say that, if they could be controlled, most of those lesions, which are now so common that few adults escape them in some form, could be prevented altogether. This is equivalent to saying that periodontia's contribution to preventive dentistry can, in its field, be made well nigh complete, providing that the knowledge already available be applied to daily practice, and the patient's coöperation be obtained. There are in fact but two obstacles to measurable success in this field—lack of interest on the part of the general practitioner of dentistry, as a result of which he fails to recognize and prevent the early manifestations of disease; and difficulty in always obtaining the coöperation of the patient in the program for prevention.

While it is possibly true that in some instances heredity, chronic systemic disease, diet deficiency, etc., may predispose to periodontal disease and unfavorably influence prognosis, it is also true that the one factor more or less common to all cases, and more directly related, is the failure to use vigorously the teeth and jaws in mastication, creating what might be called a disuse atrophy, resulting in undersized and poorly nourished tissues having increased susceptibility to disease. The logical remedy would be restoration of function through efficient use. But since there is little incentive to use either the teeth or jaws

under conditions of modern civilization, functional activity by mastication needs to be supplemented by artificial exercise, such as may be given by the tooth brush, and by Stillman's appropriately called "physical culture for the gums." Just as the athlete makes use of dumb-bells, clubs, horizontal bars, etc., in developing and making more vigorous the tissues of his body, so the patient should be encouraged to make use of artificial means, if necessary, to provide that exercise so essential to the health and well being of the teeth and jaws. It is obvious that, in this, one is dependent for success upon the coöperation of the patient, without which complete success is impossible. If there could be given to the organs of mastication, the vigorous use accorded them by primitive man, a long step would be taken in restoring to their tissues the immunity to disease which they enjoyed in him in such abundance. To recreate these conditions as far as that is possible is the first step in the prevention of periodontal lesions.

The second and equally significant factor to be brought under control is that of bacterial infection. So important is this in the etiology of chronic inflammatory diseases of the gums and subjacent tissues that it is well within the truth to say that, without the presence in the mouth of bacteria, such diseases could not occur. Among these organisms are the streptococcus and staphylococcus, both of which, but especially the streptococcus, possess in a large measure the attribute of tissue penetration. They are present in the mouth throughout life, but it is only under the condition of low cell-resistance that they are capable of invading and causing disease in the tissues that surround and support the teeth. This invasion frequently takes place long before there is any visible evidence of disease, inducing those microscopic changes in the pericementum known as rarefying pericementitis fibrosa, which undermines and destroys the pericementum, at the same time preparing the way for those grosser pathologic changes with which we are all more or less familiar, such as gingivitis, gum recession, pocket formation, suppuration, traumatic occlusion, etc., all of which depend for their existence upon an antecedent infection. In order to prevent such invasion, and so prevent disease, it is necessary, as has been said, to maintain these tissues in health as far as that is possible, through functional activity, and at the same time to keep the bacterial flora of the mouth within normal limits by the application of the principles of oral hygiene.

So much has been said in recent years about the value of oral hygiene in the prevention of dental caries, that many have come to think of it as synonymous with preventive dentistry, which is far from the truth. It is, however, of great value in the prevention of periodontal lesions, in that it is aimed at the removal of one of the two most important factors in the etiology of periodontoclasia; viz., the bacteria of the mouth. To achieve its purpose in this relationship, it is necessary that the gingival crevice, and that portion of the teeth in direct contact with the gingivae, be given first consideration, for it is at this point that the bacteria gain entrance to the subjacent tissues. For this reason, the polishing of the exposed surfaces of the teeth, so admirably done by the dental hygienist for the prevention of caries, is entirely inadequate for the prevention and treatment of these lesions. The gingival crevice throughout its circumference should be explored and its contents removed. Further than this, patients must be taught what constitutes proper care on their part and must be encouraged to persist in such care. In order to accomplish this they should be provided with brushes, dentifrices, waxed silk tape, etc., and their uses demonstrated. It is also necessary to have the whole-hearted coöperation of the general practitioners of dentistry; that is, the family dentist, to whom the patient naturally goes in the first instance, and upon whom he logically depends for the prevention and treatment of all his dental ills. With such coöperation, it is entirely possible, through the application of our present knowledge, to prevent most, and possibly all, of the lesions which tend to break down and destroy the supporting tissues of the teeth.

It should be kept clearly in mind that periodontoclasia is an infection, and one which occurs only under conditions of lowered resistance, and that the logical way in which to prevent it altogether is by keeping the mouth clean and as free from bacteria as possible, at the same time stimulating the circulation in the tissues of the periodontium by restoration of functional mastication, and supplementing this by artificial stimulation. When the dental profession realizes this, and puts it into operation in daily practice, not only will it have taken a long step in the prevention of periodontal lesions, but a most effective one in the prevention also of those twin evils, dental caries and pulpless teeth.

CARNEGIE FOUNDATION'S BULLETIN ON DENTAL EDUCATION IN THE UNITED STATES AND CANADA

REVIEW BY THE FACULTY OF THE SCHOOL OF DENTAL AND ORAL SURGERY OF COLUMBIA UNIVERSITY[1]

New York City

1. Bulletin Number Nineteen of the Carnegie Foundation was published on June 27th, 1927. It is a report to the Foundation on an investigation of dental education in the United States and Canada by Professor William J. Gies of Columbia University, based on a personal study of all dental schools in both countries, and a broad view of all dental educational agencies of the present day. The period of its preparation was about six years, and the volume consists of 692 pages of closely printed material, with a good index.

2. Following an introductory essay on the scope and purpose of modern dentistry, the work is divided into seven main parts, as follows: I. General history of dentistry; II. Present main features of dental education in the United States; III. Prospective improvement of dental education; IV. Dental education in Canada; V. General views and conclusions; VI. Dental schools in the United States and Canada; VII. Appendix, containing supplemental data of importance prior to publication. An admirable preface by Henry S. Pritchett, President of the Foundation, gives the reader a clear picture of the whole field under discussion.

3. *Part I* covers the general history of the practice of dentistry and the general history of dental education in the United States. In this section, covering about 50 pages, the historical picture is drawn in a manner so compact and explicit as to make in itself a document of permanent value to dental literature. It prepares the way to a clear

[1] Report of a special committee consisting of William B. Dunning, Chairman, Oscar J. Chase, and Leuman M. Waugh. Presented to the Faculty at a meeting held on January 19, 1928, and adopted at a meeting held on February 16, 1928. [Received for publication on February 23, 1928.—*Ed.*]

understanding of many elements in the problem of allocation of dental science in its relation to general medicine and public welfare.

4. *Part II* considers the "conditions of the practice of dentistry that determine its educational requirements—its present legal status, the types, number and distribution of dental practitioners, with a special reference also to dental service for the Negro group. The evolution of dental education is recounted from the apprenticeship system of instruction in the offices of practitioners, through the commercial and proprietary stages of college teaching, to the final emergence of the dental school as an integral part of the modern university. In this connection the history and influence of the various educational bodies, with the part that each has played in the general advance, is clearly outlined, notably that of the Dental Faculties Association of American Universities from 1908 to 1923, in its work to establish dental education on the university basis. Throughout the whole report the Director of the study has spared no pains to insist that this department of professional education is properly one of university management—an idea which, indeed, has approached so nearly to realization that at this writing only two proprietary schools remain in the United States. Following these chapters is a statement of the work of the dental schools as regulated by the state laws and by the professional organizations, present day curricula and teaching of dentistry, and the equipment and financial support of the dental schools. In this section the general progress, present status, and essential importance of dental research are put forth in an impressive and thoroughgoing way.

5. *Part III* is devoted to the consideration of the "prospective improvements in dental education in the United States." Herein is presented the fruit of the whole study. The great mass of facts having been duly evaluated in the light of the best contemporary opinion—the Director of the study having received the cordial assistance and coöperation of the leading dental educators throughout this country and Canada—recommendations are made of far-reaching importance, which will be discussed later in this paper.

6. *Part IV* covers the subject of Dental Education in Canada. *Part V* is a statement of general views and conclusions in which the conception of oral "health service" in relation to the public welfare is put forth in a manner altogether worthy of that famous phrase.

7. *Part VI* comprises 372 pages of closely printed matter giving exhaustive, detailed information admirably arranged, of every dental school in the United States and Dominion of Canada, as based upon the writer's personal inspection and inquiries. Much of the data presented was submitted to the Director by the officers of the schools visited, of which he makes due acknowledgment; but the sifting and arrangement of the enormous mass of material thus obtained is the work of one man, and it is difficult to overpraise the industry embodied in this compilation of accurate information. *Part VII*, as before stated, is an appendix containing supplemental and tabulated material, and [data on] recent developments in various schools which had undergone transition during the year 1926 just prior to the publication of the report.

8. Within the brief compass of your Committee's review, it is possible only to discuss the outstanding features of a work which may fairly be called monumental. Fortunately the debatable part is confined to a few chapters; the great part of the book deals with matters of fact, verified and re-verified by a compiler whose ability and integrity in search of truth are beyond question. The fact that this eminent service to the cause of dental education has been the work of a layman, in the sense that he is not a dentist, is indeed extraordinary except for the fact that the natural endowment and long training of a Doctor of Philosophy in accurate observation and rigorous thinking have resulted in a capacity for the severe intellectual task presupposed in the nature of such an inquiry. The comment of some critics that a serious study of dental education by one who is not a dentist, is nonsense or worse, is as shallow as it is regrettable; yet many dentists who are not willing to read the report carefully for themselves will jump to that opinion. We refer to what appears to your Committee as the very trifling "Rambling Review," so-called, of the report by Dr. C. Edmund Kells in the January, 1928, issue of *Dental Cosmos*. That a man of the well-beloved Dr. Kells' reputation should descend to a mere play-to-the-gallery in discussing a matter so serious as the advancement of learning in his chosen profession, whoever the investigator under discussion may be, is very unfortunate, and tends to confirm a suspicion that close reasoning is not an attribute one expects to find in the broad gallery to which he appeals. No intelligent reader can question

the objective attitude of the report in question. It is an impersonal presentation of a study of conditions, needs, and tendencies that exist today, from which are deduced opinions, with reasons for the same. Those opinions and reasons will stand or fall on their merits, and not because of the professional status of the proponent. Dr. Kells' critique is wholly subjective, being his own "rambling comments," which do not attempt any serious discussion. Whether or not Dr. Gies is a dentist, it is nevertheless true that his work is being studied most seriously by the leaders today in the general reorganization of dental education by American Universities; and further, that few dentists possess his knowledge of the subject as an educator, while no dentist has ever duplicated his personal observations of actual teaching conditions.

9. Without attempting to sketch in brief a line of reasoning which may be read in its entirety by all concerned, the following are the main conclusions reached by this report:

(a) That dentistry is an essential department of "health service" affecting the physical welfare of the individual and often the duration of life.

(b) Dental service demands the same educational status as general medicine as to pre-professional and professional studies, but, owing to the highly specialized procedures peculiar and necessary to dental practice, dental education should be, at least for the present, separate but closely affiliated with general medical education. Such adjustment can be achieved ideally only under university management.

(c) That the minimum pre-dental college requirement be the same as for medicine—at present two years—during which, in addition to cultural studies, special additional courses to test the vocational aptitude of the student should be required.

(d) That the professional undergraduate curriculum be arranged within three years, preferably of more than eight months each, and designed for the training of general practitioners only.

(e) One year, or more, of *graduate* work in the main departments of oral specialization, as oral diagnosis, oral surgery, orthodontia, periodontia, dental prosthesis, etc., with an advanced degree in recognition of qualification to practice these specialties.

10. The above constitutes the so-called "two-three-graduate" plan, as contrasted with the "two-four" plan of two pre-dental and four professional undergraduate years, which has lately obtained in New York State and elsewhere. A change so important is necessarily debatable—the advocates of the "two-four" plan maintaining that to adopt a three-year undergraduate curriculum would be a retrograde step. This contention is answered, however, by the proposed lengthening of the academic year and removing from the professional curriculum subjects that are proper to the pre-dental college courses, thereby concentrating the dental curriculum itself. The great advantage of the "two-three-graduate" plan is the saving of one year of the student's time in preparing for general practice—and yet providing the necessary *graduate* facilities where needed.

11. President Pritchett in his preface states: "The process of integration of the dental curriculum must obviously be one of experimentation." To what extent the recommendations of the report are sound in practice can be seen only in the long run; time will show.

Meanwhile, and within its first year of publication, a very significant change has been approved by the Educational Department of the State of New York, whereby a three-year curriculum on the basis of the four-quarter or trimester plan may be substituted for the four regular academic years.

12. While the true value of the recommendatory part of this report must await the verdict of time, we, the Faculty of the School of Dental and Oral Surgery of Columbia University, may here and now express our appreciation of the great service rendered the cause of dental education by this work of the Carnegie Foundation, and to this end your Committee proposes the following resolutions, which, if adopted, might properly be published with this review:

Whereas: We, the Dental Faculty of Columbia University, having duly considered the Report on Dental Education in the United States and Canada, to the Carnegie Foundation for the Advancement of Teaching, by William J. Gies, it is

Resolved: That in our belief, this report covers the first systematic study of its kind of national scope ever attempted, and that it comprises a body of information at first hand of fundamental value in dental education; and be it also

Resolved: That the recommendations for the reorganization and improvement of the dental curriculum are given with weighty reasons, and are worthy of most careful consideration by all dental educators; and, further, be it

Resolved: That we record hereby our sense of indebtedness to the Carnegie Foundation for this beneficent service to humanity.

METHODS FOR CONTROLLING VARIABLES IN THE CASTING PROCESS[1]

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With an Appendix by

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It is indeed a pleasure to be invited to give to this Society an exposition of a subject so vital and important to dentistry as the control of variables in the casting process. The importance of this subject may be gauged when we realize that only as we control variables in the casting process can we eliminate failure in the end products of that process.

The answer to Herbert Spencer's question, "What knowledge seems of most worth?", is surely: "That which conserves the health." And teeth play a fundamental part in this conservation. In this connection, we may as well ask ourselves this question: "Do we, as a profession, keenly appreciate the magnitude of our responsibility?" If so, we must respect the histological, physiological, and pathological attitude toward their work of such eminent authorities as Rosenow, Hartzell, Billings, Price, and Duke, at the same time that we commend the mechanical and artistic ability of such men as Tinker, Chayes, Thompson, McCullom, the Mortenson brothers, Coston, and Goslee. We must insist that our shortcomings be of the head and not of the heart, striving constantly to raise the standard of efficiency and of health service for the individual dentist, as well as the profession, and render to humanity a genuine and honest service.

Chance and change are ever busy. Certainly we have taken many chances experimenting on the value of our inlays and abutments on

[1] Read at a meeting of the First District Dental Society of the State of New York, New York Academy of Medicine, March 7, 1927.

vital teeth. Now it appears we have had an awakening, stimulating us to a fresh change, which surely must be for the better. "Man decays and ages move," writes a poet; but the great sum total of human knowledge does not decay. Rather it ever grows and develops. The torch of progress is passed on from hand to hand and the thoughts of men are widened with the march of centuries. Obviously then, research, knowledge, and teaching are the stepping-stones on which the profession must continue to build. Dr. Taggart was perhaps the first man fully to realize this need, and to pave the way toward the attainment of our present state of proficiency. He, the pioneer, has brought the casting process to a point from which perfection is in sight. And the profession must further promote this effort even at the cost of self-sacrifice. We must be willing to face, if necessary, the scrapping of our old ideas and cherished methods. When we regard our past mistakes and their consequences, we may well have a stricken conscience. But this, uncomfortable as it is, may lead to dental progress, creating action and stimulating advancement.

Our profession is not alone in darkness, for other callings also sense their shortcomings. Education has become the chief need of the world—its one holy cause. Knowledge must henceforth be the light, the guide of mankind. More of it must be sought from the original sources—man and Nature. It must be even more widely diffused; there must be an absolute freedom of both research and teaching. There must also be re-education of the soul and of the heart as well as of the intellect. Ideals of service must supplant those of selfishness and greed. Nothing else can save us. I shall live, and I hope to die when my time comes, convinced that this goal is not only attainable—in our peculiar case, a perfect gold inlay—but that we are, on the whole, with however many retrogressions, making progress surely, if slowly, toward the light. We suffer from the lack of any adequate body of scientific research. We need, for our special problem, greater knowledge of metallurgy, of shrinkage and expansion of waxes, investments and golds, as well as a wider knowledge of biological laws, as they relate to the teeth and the mouth. Yet, in spite of these handicaps, I feel certain that with me, at least, the "law of compensation" is always at work. Injuries to patients, unavoidable because

of our inadequate knowledge, have in the end been a source of stimulation and of consequent benefit to others. Perhaps before many years the combined efforts of research workers and earnest practitioners will render further experimental work on patients avoidable.

Often when I consider that, since 1907, I have tried to get a perfect gingival margin, my energies seem wasted, my efforts fruitless, and love's labor lost. I have not realized my expectations nor succeeded in capitalizing my ideal. The goal has not been attained. The casting process, with its numerous failures over a period of many years, has experienced disaster and is in great danger, especially since the various methods of repair are at war with each other instead of coöperating to stem the rising tide of failure. Crowns, inlays, and three-quarter veneers have suffered considerable criticism, and justly so. This uncertainty today breeds discontent everywhere among the profession, as well as the laity. Therefore, an earnest effort to standardize the methods for the control of variables capable of jeopardizing the casting process should be made by every dentist.

Unfortunately, we have been unwittingly misled since 1907 by many earnest, honest, and conscientious members of the profession, by many successful commercial institutions, as well as by our own individual efforts, study, and research, in attempting to arrive at the truth and perfection of the casting process. At the same time we have sincerely hoped that these combined endeavors would ultimately enable us to perform so responsible a service—where indicated by ravages of decay and the need for bridge abutments—in such a manner that no harm should follow to the teeth or to the organism as a whole. The variables, nevertheless, have materially jeopardized our serious efforts. The finished product has fallen far short of the high degree of perfection at which we have constantly aimed; in other words, we have heard the music and missed the tune. Yet in face of these difficulties and imperfections, the profession since 1907 has constantly, untiringly, and conscientiously explored every avenue promising to lead to its desired goal. We have tried out various ways of cavity preparation, different kinds of waxes, investments, and surface agents; many types of casting machines; various methods of melting different kinds of gold and platinum combinations; different furnaces and devices for wax elimination, consisting of the carbonization and

the so-called quick method without carbonization; casting in a hot mould then a cold mould; and, finally, the indirect method with its many different kinds of swedges and materials for dies; also the sledge hammer, so ably and efficiently demonstrated by our good friend and coworker, Dr. W. Coston, who recognized since 1907 that, with the direct method, large castings could not be made to fit. To cap the climax, an inlay cement, the color of gold, was produced to hide effectively our defects—and all this is called efficiency.

Most of us were warmed by the enthusiasm of discovery and stirred by the stimulus of new ideas when, in 1907, Dr. Taggart gave us the disappearing pattern and the casting process. In 1910, however, our cat had its fur rubbed the wrong way by Dr. Hunter of London, when he wrote his paper on sepsis and anti-sepsis in surgery, in which he called attention to America's criminally unscientific dentistry. Unfortunately, and undoubtedly for good reasons, this arraignment was not published until July, 1918, in our *Dental Cosmos*.

As our study, research, and teaching have up to the present time been unable, through preventive means, to check the ravages of decay or materially to assist nature in maintaining a healthy investing tissue, it undoubtedly is obvious to all that it is still imperative that we, as a profession, continue our efforts to perfect our mechanical technique. Furthermore we must unify and epitomize our deductions to that definite end whereby it will be possible for us, as conscientious practitioners, to render suffering humanity a real service in the form of alleviating pain, restoring lost tooth structure, replacing missing teeth, and teaching patients how to keep twenty-eight inches of investing tissue in as high a state of health as possible. And we must always keep in our consciousness, as Dr. C. N. Johnson has well stated, the one outstanding fact: that prevention is the heaven of the future wherein hangs the rainbow of promise. Pity the man who has no such vision looming large on the horizon, for daily are we impressed by medicine and science with the important part which dentistry should play in maintaining health, and quite as much are we impressed by the inadequacy of our present methods. However, to be specific relative to our subject, it is the unseen, unknown factors, elements, or obstacles that apparently jeopardize our final results in bridge abutments, crowns, and inlays. This is my real reason for accepting your

committee's kind invitation to appear before you today and to read a paper on this subject.

So much has been written on cavity preparation that we sincerely hope this most fundamental requisite is thoroughly appreciated and mastered by the profession. With this assumption we shall be free to treat only such phases as are apparently the cause of our failures in the casting process. The first and most important of these is the contraction and expansion of our waxes.

Undoubtedly, the variables are directly responsible for (1) the deficiency of gingival margins, the most vulnerable area of individual crown veneers, and inlays; (2) shrinkage of buccal, lingual, and occlusal margins, inviting leakage, discoloration, decay, and exposure of the pulp; (3) fracture of cusps in attempts to seat castings—not in evidence at the time, but later through seepage and experience of the fractured cusp or cusps by the patient; (4) strangulation of living dentine cells, due to shrinkage in full and veneer crowns, many times causing death of pulp; (5) trauma, by driving casting to place, causing pericementitis and untoward sequelae; (6) ill fitting abutments or crowns, causing seepage, decay, and loosening of bridges, jeopardizing the priceless remaining teeth, and often helping to encourage devitalization, which frequently results in abscesses (these may become foci of infection, which in turn may undermine the health, and finally pave the avenue for dentures); (7) gingival shoulders of tooth structures which, through shrinkage, are not reached by the periphery of the casting, and may, during attempted removal, lacerate investing tissues, causing inflammation, recession, sensitiveness, decay, and often exposure; (8) serious expansion of tooth walls during setting, through the use of a medium mix of cement (two things cannot occupy the same space at the same time), causing enamel margins, which before setting were flush, to project beyond the gold. To deny that these failures are as great as has been suggested is to deny the matter of our common knowledge, experience, and observation. It must be evident from the shortcomings listed above that the reasons for these failures can be eliminated only through a better understanding of chemistry, physics, and metallurgy—a more scientific knowledge of the materials entering into the equation. Our great need is for some pioneer of science to precede the rest of us into new fields of endeavor,

and lead the way to the fulfillment of our most imperative requirements.

To those seeking individuality and distinction, without extravagance in dental castings, I suggest Dr. Van Horn's paper;[2] also Dr. Meyer's paper.[3] I am frank to admit that I have nothing to add to their early research and experiments, except (1) the suggestion of quick elimination, requiring only 20 to 22 minutes for investing, setting, and the elimination of wax, and (2) casting into a hot mould. After reading these articles, one may be sole judge of his own results, appreciating the fact that the status of an individual dentist is measured by his ability to do things correctly without supervision; and that by our works we shall be known.

Closely related to our subject is a consideration of the principles governing crown and bridgework. Ever since the exigencies of accident or disease have caused the loss of teeth, the individual suffering from this loss has endeavored to supply, or to have supplied for him, the missing members. Undoubtedly, a greater amount of ingenuity has been expended in bringing restorative dentistry to its high attainments than has been elicited in any other branch of our calling. Nevertheless, the replacement of missing teeth by means of bridgework of any type is always a problem of serious portent and one still far from its solution. This is true, obviously, because the remaining teeth or environmental tissues, or both, must of necessity assume the burden of restoring the function of the missing unit, or units, and this responsibility must be assumed *in addition* to the requirements incidental to their normal activities.

Dr. Ash of New York says: "There is but little variation from case to case in the ability of the alveolar ridge to support occlusal stress, transmitted through a restoration, except the tendency to gradual settling. But there is wide variation in the ability of abutment teeth to support, or even to retain either removable bridges or partial dentures. Briefly stated the principal factors which govern the ability of abutment teeth to perform the duties which we may wish to assign to them are, the load, the length of roots, and extent of their

[2] Van Horn: Casting; a review and commentary. *Dental Cosmos*, 1910, lii, p. 873.

[3] Meyer: Explanation of, and partial remedies for, some of our failures in cast-gold inlays. *Dental Cosmos*, 1926, lxviii, p. 525.

alveolar support, and the degree of tipping, if any, of the root from its normal position. No mention is made of periapical infection, as it is assumed that any infection found will be cleared up by treatment, or the tooth extracted." Personally, however, I am with Dr. Rosenow of Rochester: "Why let sleeping dogs lie, especially under bridges?" It has been my experience, agreeing with the above, that you cannot promise immunity to any patient with restorations or non-vital teeth longer than twenty-four hours. Further quoting from Dr. Ash: "By the term 'load' we mean the amount and direction of stress which the abutment teeth will be called upon to support. This varies with the shape of the ridge and the density of the mucosa, also with the extent of the appliance. It goes without saying that the longer the root the greater the ability of the tooth to support stress, assuming that the surrounding bone is healthy." Therefore, it will be seen that the condition of underlying tissues is the all-important consideration in fixed and removable bridges. In many cases the bite must be opened, with due consideration given the balancing of occlusion, the spherical theory, and Gysi's Gothic Arch.

Since the health of our patients is the fundamental and first consideration, I am of the opinion, as stated above, that the removal of all foci of infection is of paramount importance. Following this, the investing tissues of all remaining teeth must be placed in a state of health. This gives the dentist a fearful responsibility. What is left after the removal of all possible foci of infection becomes sacred ground. The proper treatment of remaining teeth is of vital moment to the patient, involving not only his oral but his general health.

Fixed bridgework has suffered from empirical methods applied indiscriminately by many members of our profession. Possible injury to the supporting natural teeth and the requirements of hygiene and sanitation have been largely ignored. The conservation, promotion, and maintenance of health, the fundamental requisites in the replacement of missing teeth by any method, have been largely ignored. Faulty occlusion plays a most prominent part in the exfoliation of either individual or abutment teeth. Stress in a direction and of a degree which the tooth is by nature wholly unprepared to meet is the hardest element to deal with in either fixed or removable appliances. If we could construct and antagonize our restorations so

that the teeth would not be moved in function, the intention of nature would be more nearly fulfilled. I have no pet theory of articulation and occlusion to promulgate, nor is this paper intended to be an exposition of any panacea for our troubles. But so important is the proper relation of the maxillary and mandibular teeth to each other that I feel justified in saying that the future of dentistry in all its branches depends on the solution and understanding by the rank and file of us of this serious problem. Therefore, it seems imperative to acquire good study models of low-fusing metal accompanied with correct check bites, mounted upon an articulator that will exactly duplicate all of the movements that the individual has acquired. The various excursions, or range of occlusion by the Gothic Arch tracings, as suggested years ago by Dr. Gysi, must be ascertained and the models constructed to follow. Agreeing with Dr. Tinker, after experiences extending over a period of twenty years, I have found it safer to construct cusp relation in harmony with the condyloid path present in the individual case, than to establish a new occlusion arbitrarily.

First, we recognize the necessity for analyzing the conditions found in the mouths of those requiring bridgework. These include the stability and number of possible abutment teeth, and the character of the soft tissues and alveolar ridge. From the diagnosis thus made, we can arrive at a decision as to the type of restoration to be employed. The stability of an abutment means virtually its ability to support, or retain, a fixed or removable restoration. The items governing this are the load, the length of the roots, the extent of their alveolar support, and the degree of tipping, if any, of the root from its normal position. Every replacement must be one of three: first, tissue bearing; second, tooth bearing; third, composite, i.e., both tooth and tissue-bearing.

To come down to the present time, it is a question—considering the opportunities of the present generation, embracing both modern knowledge and the experience of those who have gone before, and also considering the number of those who are doing the work now as compared with the number of those who did it in the past—whether we have any cause to flatter ourselves on our progress; whether, taken as a whole, the work of the present is much of an improvement over that of a quarter of a century ago. Certainly, a very large percentage

of the work done at present is no whit better, and much of it is worse, than that done twenty-five years ago, and does far more harm than good. There is now a realization that no one person can in a lifetime solve the dental problems and produce a device for replacing all types of lost tooth areas.

Realizing that a bridge is no better than the abutments which support it, it becomes imperative that each casting, whether crown, three-quarter veneer, or inlay fits properly—in other words, so well that no cement is visible after setting, especially at the gingival margin, and without expanding the tooth walls or impinging too hard upon the exposed dentine. To accomplish this, castings must duplicate the wax patterns at the time they were made; and here begins the control of an important variable, namely, the wax itself. Dr. Fred Meyer of Minneapolis proved in 1912, and again with me as a spectator in 1924–25, that for every 5 degrees of temperature change on the downward scale there was approximately 1/1000th of an inch shrinkage, or 0.1 per cent. Therefore, if a pattern was made at 95 degrees and invested, using tap water at 50 degrees, there would be 45 degrees of difference in temperature, or 9/1000 of an inch in shrinkage. To this add the shrinkage of gold, which is 1.4 to 1.7. If a wax cylinder about one inch in length and about one millimeter in thickness is invested and cast, using the above method, it would shrink from 18/1000th to 22/1000th of an inch, depending upon the technique of handling and the method of casting. Permit me to call attention to the figures of a few simple experiments Dr. Meyer and I made in January, 1925.

1. A wax cylinder was prepared, 3/16 inch in diameter and 1.0011⅓ inches long, at room temperature, 72°F., with round platinum caps tacked at each end, to insure better measurements. This wax cylinder was invested, using tap water at 55 degrees, allowed to set one hour, burnt out in an electric furnace at 650 degrees for 30 minutes, and cast cold in a pressure machine using 5 lbs. The measurement of the gold cylinder was 0.9890 inch; it, therefore, had a shrinkage of 0.022⅓ inch.

2. Wax cylinder measured 1.008.
Gold cylinder measured 0.988–0.020 shrinkage, or 2 per cent.
Water used at 69°F.
Room temperature—78°F.

3. Wax cylinder measured 0.993 at 60°F.

Invested at 100°F. Allowed to set 45 minutes in heater at 125°F., then heated in a furnace 35 minutes at 6 to 650 degrees. Cast in cold mould, using 4½ lbs. pressure. Measurement of gold after casting: 0.985–0.008 shrinkage.

4. One measured at 60°F., invested at 115°F. The shrinkage after casting was 0.005–0.5 per cent.

If heat were used up to 130°F., to expand the wax, and cast in a hot mould, the shrinkage would be nil.

These experiments show that in order to duplicate the patterns made at body temperature, they must be expanded to compensate for the shrinkage of the wax, gold, and the investment with its porosity. This is readily accomplished by having the armamentarium and materials hot; namely, the base or sprue former, the ring, bowl, spatula, and brush in really hot water, or an oven about 130 to 140°F.; the dry investment must likewise be warm, with hot water used for mixing. After investing is complete, it must be placed, while setting, in a container that will register 130 to 135°F.; the wax should be allowed to assume the temperature of the investment during crystallization, thereby getting maximal expansion of wax without distortion.

The best linear measurements of a casting have been obtained by using the following method of investing. Take Dr. Knapp's No. 1 investment and coat pattern about ⅛ in. thick. Immediately mix Taggert's investment according to directions, and pour over Knapp's or first investment. Place in container or oven at 130 to 135°F., allowing about 30 minutes for setting. Then eliminate by the quick method utilizing from 5 to 7 minutes, and cast hot with about 10 lbs. pressure. One convincing experiment is pursued as follows:

Make a steel first-molar tooth according to Black's measurement, and prepare in same a typical M.O.D. inlay cavity. After completion, case-harden this and endeavor to make an inlay, disregarding the control of wax, shrinkage of gold, and investment, also casting into a cold mould—in other words, using the method almost universally employed today in the profession. After casting this, attempt its seating, permitting the use of steel instruments, mallet and anvil, and note the results after it apparently is driven to its destination. It will be found that the inlay only approaches its destination by two-

thirds or three-quarters of the distance, conclusively proving to ourselves that with the old methods and conception of technique, it is a hopeless undertaking. When you have satisfied yourself relative to the impossibility of accomplishing this by ordinary methods, make another, incorporating into the technique the suggestion of Dr. Van Horn and Dr. Meyer, and you will note that the inlay may be seated with thumb or finger pressure.

I feel confident that if practitioners will work out the above outlined simple, economic, sufficiently elastic and practicable technique, they will find that duplicating wax patterns in gold is a genuine joy. With one hand, we must hold fast the best we now possess and, with the other, reach out and grasp all newly advanced suggestions that have merit.

To summarize: up to the present time the greatest percentage of castings have been extravagantly and conscientiously conducted to supposed completion, with little advancement during nineteen years toward a perfected ideal, due particularly to a lack of knowledge of the chemical, physical, and metallurgical properties of the materials used. Obviously, in spite of all its shortcomings, the casting of inlays, three-quarter and full crowns is here to stay; but our only salvation lies in an immediate, full, and conscious realization of the scientific control of variables, if we expect accurately to duplicate our wax pattern.

SERIOUS SHORTCOMINGS ENCOUNTERED IN THE PAST

1. Deficiency, particularly at the gingival margins.
2. Shrinkage, which did not enable the casting to reach its destination.
3. Expansion of tooth walls, endangering the pulp, with possible fracture, often not noted at the time of setting.
4. Prevention of proper setting, with the cement as a factor.
5. Trauma, in attempting seating.

The above difficulties may be due to:

1. Improper cavity preparation.
2. Shrinkage and expansion of wax.
3. Shrinkage of investment.
4. Shrinkage of gold.
5. Difference in shrinkage of castings, due to different shapes of cavities.

3. Wax cylinder measured 0.993 at 60°F.

Invested at 100°F. Allowed to set 45 minutes in heater at 125°F., then heated in a furnace 35 minutes at 6 to 650 degrees. Cast in cold mould, using 4½ lbs. pressure. Measurement of gold after casting: 0.985–0.008 shrinkage.

4. One measured at 60°F., invested at 115°F. The shrinkage after casting was 0.005–0.5 per cent.

If heat were used up to 130°F., to expand the wax, and cast in a hot mould, the shrinkage would be nil.

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4. Shrinkage of gold.
5. Difference in shrinkage of castings, due to different shapes of cavities.

6. Improper setting.
7. Ineffective finishing of casting, in and outside.
8. Overheating of the gold; therefore, too much oxidation.
9. Casting into cold mould with consequent impaired linear measurements.
10. Mixing investment too thin, causing additional shrinkage.
11. Too much heating of mould, or investment, causing additional shrinkage.
12. Failure to consider proper casting pressure, usually 10 or 12 lbs. where quick elimination methods are used.

Relative to the deductions just cited, it becomes imperative that we (1) become more saving of vital tooth structure; (2) substitute harder gold alloys and less bulky castings for softer gold alloys, bulky inlays and attachments, thereby saving more pulps; (3) improve our wax patterns at the gingival margins where they usually fail to fit owing to shrinkage of wax and gold (certainly the casting will be no better than the wax pattern); (4) seek to expand our wax patterns as much as possible without distortion, to compensate for shrinkage of wax, gold, and investment; (5) adopt a technique and investment that will give the least shrinkage in wax elimination, also produce the smoothest casting; (6) mix investment as thick as it can be successfully handled; (7) adjust castings to prevent their impinging on cavity walls, especially the living dentinal fibrillae, thus avoiding a tendency to strangulate the pulp, preventing fracture, and permitting space for cement; (8) cast into a hot mould in place of a cold mould to obtain better linear measurements; (9) have the cast heated to cherry red, dropped into sulphuric acid to remove oxides, pickled in hydrofluoric acid to cleanse thoroughly, and examined with magnifying glass for the detection and removal of all globules caused by bubbles in the investment; (10) make proper adjustment of the casting to the tooth; secure an effective finishing, including proper sulci, grooves, cusps, contacts, embrasures, and occlusion and articulation, coupled with an efficient method of setting.

You will agree with me, after this enumeration, that it is difficult for one not in touch with recent developments and progress in the field of casting to follow the lines of argument and research in recent stupendous discoveries. Yet these principles throw much clear light

upon the mysteries of the process which, obviously, is much more complicated than it has been thought to be. Deep calleth unto deep here; and it is far from likely that we have yet plumbed the abyss.

In conclusion, I wish to say that if I have contributed anything by verifying the timely and noteworthy research carried on by Drs. Taggert, Van Horn, and Meyer, I shall feel amply repaid for my endeavor. I trust you will realize that my mistakes are of the head and not of the heart. I would also have you realize that we all invite constructive criticism. I appreciate, on my part, that you are valuable members of society and of your profession, imbued with a spirit of responsibility toward humanity, seeking to raise the standard of the profession and of human health efficiency, and that you have a wholesome concern for the salvation of the social group and a watchful eye for the happiness of the present life. For these great ends are coming into the minds of men as their most satisfying possibility emotionally, into the actions of men as the greatest possible work for true democracy. To do our part in realizing these is to render genuine social service. To do less than this is to fail.

Appendix

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Wax expands on heating and contracts on cooling. These are well known facts. This expansion or contraction amounts to about 0.1 per cent for every five Fahrenheit degrees of change in temperature of the wax, more or less depending upon the wax used. These changes cannot be disregarded if we wish to employ the material to best advantage in our work.

Plaster of Paris, we are told by Dr. Harder of the University of Minnesota in the 1926 December issue of the *Dental Cosmos*, expands slightly on heating but contracts from its original volume—sometimes as much as 1 per cent upon being cooled after it has been brought to an excessive heat. Plaster of Paris is an important ingredient in all our investment compounds. These are facts also that cannot be disregarded, if we wish to employ this material in our investments.

All alloys of gold in our use shrink as they pass from the molten to

the solid state. The shrinkage is about 1.5 per cent, more or less depending upon the alloy used. These are facts also that cannot be disregarded, if we seek the best results in our work.

For the last twenty years all the above facts have been disregarded by the majority in the casting process. And what have been the results? Large inlays and crowns that can never be properly seated. Wide-open margins that harbor decay when the cement has washed out. Uncovered gingival margins due to mesio-distal shrinkage. Compression of the fibrillae of the dentine, causing soreness of the tooth and often death of the pulp. Bucco-lingual expansion of the tooth, often causing fracture of the buccal or lingual cusp. And all the annoyance and trouble that goes with trying to fit something that does not fit.

Step by step, I have worked out a technique in which we can utilize to our advantage some of the above factors or forces which have always worked to our disadvantage. But, in order that you may understand the reasons for every step in this technique—that you may make the best possible castings with the fewest failures—it will be necessary for me to enumerate certain fundamental principles and explain them in detail.

1. The thinner a wax model, the less shrinkage of the gold casting. As the gold passes from the molten to the solid state, it must reach a certain state of solidity before it is able to compress the investment and shrink in spite of it. The thinner the bulk of gold, the nearer to the solid state it must be before it can compress the investment and shrink in spite of it. Consequently, the least shrinkage is registered between points that tend to bind. Conversely, the bulkier an inlay, the more it shrinks between points that tend to bind.

2. The thinner the occlusal portion of an M.O.D. inlay in proportion to the mesial and distal portions, the less shrinkage from mesial to distal. If the occlusal portion is thin, and the mesial and distal portions are bulky, the occlusal portion will begin to congeal and shrink before the mesial and distal portions. It will complete a great part of its shrinkage before the mesial and distal portions are hard enough to overcome the resistance of the investment. Hence the inlay will register less shrinkage from mesial to distal. The converse is true, if the mesial and distal portions of the inlay are thin and the occlusal is bulky.

3. The more the wax model is chilled while held in position in the cavity, the less binding in the cast-gold inlay. When chilled, wax tends to shrink, but it cannot shrink where the shape of the cavity is such that the tooth prevents it. In such a case there must be a rearrangement of the molecules of wax to conform to conditions, or an elastic tension is established with a tendency to contract when the model is removed, or both. The former takes place largely in the early stages of chilling, while most of the latter occurs when the wax becomes fairly hard. This elastic tension is largely removed by softening the wax with a hot instrument between points that tend to bind. If the chilling is carried far enough with a large wax model on a metal or porcelain tooth, the wax will check when the tension becomes too great.

4. The warmer the investment when it sets, the more expanded the wax model. This expansion in turn compensates in a measure for the shrinkage of gold.

5. The larger the sprue hole, the longer it takes for the gold in it to congeal and the more gold fed through it before the gold casting passes the mushy stage. Consequently, the denser the casting and the less it shrinks. This, I believe, is the main advantage of the centrifugal machine over the pressure machine.

6. The shorter the sprue hole, the longer the gold in it remains molten and, consequently, the more gold fed through it before it congeals. Hence, the less the shrinkage.

7. The better the ring fits the crucible former when using warm investment, the less the danger of distortion of the wax model. Theoretically, no water or investment should be allowed to escape from the base of the ring, causing a settling of the investment, nor any movement between the ring and the crucible former in handling. This is best accomplished by a ring fitting into a groove of beeswax on the crucible former, and fitting so closely that the crucible former can be picked up by taking hold of the ring.

8. The thicker the investment when mixed, the denser and harder it becomes and the longer it resists the pressure of the cast gold as it passes from the molten to the solid state. Hence, the less the shrinkage in the cast gold inlay between two points that tend to bind.

9. The more plaster contents in the investment, the harder it

becomes and the more it resists the pressure of the congealing gold as it shrinks; hence, the less shrinkage.

10. The less heat the mold is subjected to when eliminating the wax and the moisture, the harder and denser it becomes, the more it resists the pressure of the congealing gold, the less the shrinkage of the gold, and the smoother the finished casting.

The above fundamental principle; namely, that the harder investments of a high plaster content cannot be heated to a high degree without destroying their life and resisting qualities, causes a critical stage in the wax eliminations. With large gold castings it is very difficult to eliminate all the wax without danger of overheating the plaster. The percentage of moisture in the mold is not always the same, nor is the amount of wax to be eliminated, nor the gas pressure. A great share of this difficulty can be overcome by eliminating most of the wax, or all the wax, before subjecting the mold to the flame, and by bringing the mold to the same point of saturation with moisture for all castings. This is done by a very simple device consisting of a small receptacle containing water sealed in by the base of the mold, and so arranged that the water when heated must be forced through the mold, carrying the wax with it.

11. The less foreign matter, which might have been incorporated in the wax, in the mold, the smoother the casting. If the wax is forced out through the sprue hole in a softened mass without being allowed to soak into the investment, all particles in the wax which might be left as a residue in the carbonization method are likely to be carried out with it.

12. The denser and harder the investment, the greater the pressure necessary to force the air out of the mold and the gold into it. Here is where the pressure machines have the advantage over the centrifugal.

13. The denser and harder the mold, the greater the pressure it will stand without injury in casting against it.

14. The greater the pressure in casting, the more gold fed into the mold while the gold is congealing and, consequently, the less the shrinkage.

15. The larger the button of excess gold in proportion to the size of the castings, the longer it remains molten. Hence the more gold fed into the mold before the casting congeals, and the less the shrinkage.

16. The sooner the gold is cast after being brought to a state of fluidity, the smoother the casting and the better the condition of the excess gold for future casting. Overheated gold not only oxidizes to excess, but tends to burn the plaster and destroy the smoothness of its surface.

17. The lower the karat of the gold, the lower the temperature at the melting point and consequently the less the shrinkage.

18. The less the inlay binds or compresses the fibrillae of the dentine, the less the danger of fracture of the tooth, soreness, or death of pulp.

All these fundamental principles could be considered at length, but since most of them were discussed in my paper in the *Dental Cosmos* of February 1926, and they all constitute common knowledge in a condensed form, it will not be necessary to do so.

Briefly, my technique for investing a wax model, and eliminating the wax and the moisture from the mold, is as follows:

Fill the groove on the crucible former with beeswax. Then insert a little beeswax into the sprue hole and insert the sprue supporting the wax model. This will prevent the wax model from moving when being painted with investment. Place about a cubic inch of Meyer's investment at the right end of a large glass slab. Fill the large end of Taggart Scales with Taggart investment, and balance with warm water.

Now note this very carefully: If the wax model to be invested is for a full crown, use the water in the bottle in the wax expander at about 135°F. If the wax model is for a bulky M.O.D. inlay, dilute the water to about 120°F. If it is for a thin M.O.D. molar inlay, an M.O.D. inlay for a bicuspid, an M.O. or a D.O., any small inlay or a three-quarter crown, do not use water over 100°F. Make the mix in the small bowl fitted to the wax expander. Stir enough with a spatula to see that the mix is of the right consistency—a heavy cream—and complete the mixing in the recess provided for the bowl in the wax expander. Leave the whip-mixer in the bowl until the wax model has been covered with the inner investment. From a water syringe place a quantity of water on the glass slab covering a space about the size of a dollar or a little more. With a spatula make a mix of water and Meyer's investment of such a consistency that it can be piled up fairly

high without flowing. Draw off about a third of this, and add to it more investment powder until it is as heavy as you can conveniently paint on your wax model. After painting a little of this on the cavity side of the wax model, rasp the crucible former with a file. Paint on more of the heavy investment and rasp again. This causes the heavier particles of the mix to settle, producing a dense surface next to the wax model. It also causes the bubbles and water to rise.

Then paint the rest of the wax model with the thinner inner mix, covering it to the thickness of about $\frac{1}{16}$ to $\frac{1}{8}$ inch, or a little more, and fill in clear down to the crucible former. With a rotary motion place the warm ring taken from the wax expander into the groove on the crucible former. The beeswax in the groove will seal the joint. Make a couple of turns of the whip-mixer and pour the outer investment.

Now note this carefully: If the invested model is for a crown, place it on the grate in the wax expander. If it is for a bulky M.O.D. molar inlay, place it on another ring in the wax expander. If it is for most any other small inlay, or a three-quarter crown, place it on top of another ring in the wax expander with the door slightly open, or on the top of the wax expander. Always remember this: the thinner the wax pattern, the less the necessary expansion. This is especially true with three-quarter crowns. Allow to set about forty minutes before eliminating the wax and the moisture. I time this with my x-ray timer.

When set, remove the mold from the wax expander and level off the closed end. Remove the crucible former and sprue. See that the rubber washer is in place in the lower end of the mold compartment of the wax eliminating boiler. Fill the boiler with warm water and place the mold with sprue end up in the upper compartment. Hold the mold tightly against the rubber washer and empty out all surplus water. Screw on the top part of the boiler so that it holds the mold tightly in position against the rubber washer. Place the boiler in the electric eliminator, or any other eliminator serving the same purpose, and leave until the water boils over and no more wax emerges from the sprue hole. Then remove from the electric eliminator and allow the water to continue to boil for a minute or so. The whole elimination process should not take over five or six minutes.

Turn the boiler upside down and cool under tap water. Remove the mold and place it in the electric eliminator with the sprue-hole up. *Remember this also:* if a full crown or a three-quarter molar crown is to be cast, leave the mold in the eliminator until all the moisture has disappeared from the sprue hole as shown by holding a mirror over the open end of the mold. If the crown is exceptionally long, leave it a minute or so longer. There is a good scientific reason for this procedure. I believe that most of you have cast crowns when flaw holes appeared in the occlusal portion. Close analysis will explain the reason. When you are eliminating either the wax by the carbonization method, or the moisture alone by my method, it takes longer for the heat to reach the end of the core inside the crown than to reach any other part of the mold. Therefore, it follows that if any of the wax, or moisture, in the mold fails to be eliminated, the failure will be at the end of this core or on the inside of the occlusal portion of the crown. When the molten gold makes the contact, with wax or moisture, there is a sudden expansion of gas produced by the heat from the molten gold, causing a hollowing out of the casting and sometimes a hole clear through the occlusal.

Practice has shown that by lengthening the time of elimination, in case a crown is to be cast, you will to a large extent obviate the above trouble. If an inlay, remove the mold from the eliminator while still a quickly disappearing cloud appears on the mirror when placed against the open end of the mold. The elimination of the moisture should take from sixteen to twenty minutes, varying, of course, with a slight change of temperature of the eliminator and with the size and shape of the wax pattern. Before casting, place the mold in the wax expander for from three to five minutes to allow any trace of invisible smoke or vapor to escape.

If this process is carried out carefully, one can cast an M.O.D. molar inlay or a full shoulder crown, smooth inside and outside, that will go clear to place with finger pressure on a porcelain model *every time*.

A WORKABLE VIEW OF FUNCTION IN DENTURE PROSTHESIS FROM THE STANDPOINT OF HEALTH AND PREVENTION[1]

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I. INTRODUCTION

A few months ago I had the opportunity to spend several hours discussing certain aspects of denture prosthesis with a brother practitioner and friend, who is closely connected with and is intensely interested in dental education. Our discussion gradually drifted along the line of maintaining both health and function in the oral cavity of denture patients, and also, how to proceed to attain this as our object. To my surprise this angle of denture prosthesis was entirely new to him. Briefly, this is what I gathered from him as his conception of the prevailing opinion among dentists about prosthetic dentistry, and its place and importance in the profession: "prosthetic dentistry and mechanical dentistry are considered both as one and the same thing. Prosthodontists, as a rule, are little interested in *function* from the standpoint of *prevention*. The soft tissues of the mouth, the underlying bone, and the teeth are taken and considered simply as the means of support for dentures, and that tissue destruction, bone resorption and the loss of teeth are inevitable."

This indictment of prosthetic dentistry, based on generalizations without adequate evidence to support it, is unjust. I feel, therefore, as duty bound to my specialty, and to dentistry in general, to try

[1]The first of several projected papers on various aspects of this subject, to be published in this *Journal*.—[*Ed.*]

to disprove this accusation by presenting briefly my views, and the prevailing views among prosthodontists, of denture *function* as a means of promoting and maintaining health in the oral cavity—in other words, the professional side of our specialty.

Perhaps my friend was justified to some extent, in his conclusions, because we have not been blameless. Our clinics have stressed the mechanical element in denture work to a point where the aim and purpose behind a given technic has been completely lost in the labyrinth of mechanical details. I also feel that we have too many typodont and table clinics instead of actual demonstrations, with patients, emphasizing the need for better procedures from the standpoint of health and prevention. In partial denture clinics, for example, so much time is being spent talking about the various types of clasps, the materials needed for their construction, the metallurgical aspect of the behavior of these materials, casting technics, stress breakers and indirect retainers—and furthermore, so many highly polished dentures, each of a different and intricate design, are being exhibited, —that the attending dentists justly receive the wrong and lasting impression that modern denture prosthesis is purely *mechanics*. And that good dentures, either full or partial, can be *designed* by any good laboratory or technician.

Preventive measures in dentistry in recent years have gone far in maintaining oral health, preserving the teeth and tissues in a healthy state. Periodontists throughout the country have succeeded in bringing under control periodontal lesions even in advanced types of periodontoclasia. Whether the retention of teeth in the mouths of patients past middle life, on a foundation of weakened periodontal tissues is wise or not, is a different matter. But the fact remains that we are called upon more and more to restore masticatory function for patients in this class, who have held on to the last tooth, in dread of artificial substitutes. Again the amount of tissue and bone destruction, which at present accompanies the extraction of a large number of long retained non-vital teeth, is greater than it was a few years ago, when extraction was the only logical remedy for an aching tooth. These patients present a sorry condition, with the ridges completely gone or badly mutilated, and the soft tissues weakened to a point where there is little likelihood for bone and tissue regeneration.

All denture cases, therefore, that come to us, bring with them their individual peculiarities and problems. Some of them are trivial and others are quite serious. In either case, these peculiarities and problems must be met in a concrete form, in the shape of dentures functioning efficiently, increasing function where it has been impaired, and establishing function where it is lost. And in both instances the underlying principles of design and construction are fundamentally the same. By function I do not mean masticatory function alone, at the expense of the remaining teeth or the supporting tissues; but function all along the line. That is:

First, establishing efficient masticatory function;

Second, providing and maintaining normal function in the remaining teeth, if there are any present; and

Third, stimulating normal physiologic function in the supporting tissues by means of dentures, so as to keep them in health.

II. PARTIAL DENTURE CONSIDERATIONS

We often hear that the partial denture is nothing more than a forerunner of the full denture, distinctly implying that no improvement in our methods can prevent the partial denture from being the most temporary sort of makeshift. If this were true, it would be a waste of time to discuss partial denture procedure, and, in fact, it might even be a waste of time to construct one. Fortunately, however, this is not true, and the universal interest in partial dentures proves that few, if any of us, really believe this to be true. The fact that so many of us flock to certified laboratories for certified partial dentures, although pitiful, is still an encouraging sign. It is estimated that a good denture, at its best, is only 30 per cent as efficient as were the patient's own teeth. Yet none of these laboratories can *design* a denture that will meet all of the requirements of functional efficiency. Because a partial denture is comfortable and looks well, providing teeth in good centric occlusion, is no indication that it is efficient. If the results of *unintentional* partial denture malpractice were immediate, many of us would be compelled to quit dentistry or stop making dentures.

Let me state in this connection that I do not wish to be misunderstood. My reference to certified dentures does not imply that I

consider laboratories, as a whole, unfit to perform useful mechanical work for us. What I deplore and condemn is the fact that we *demand* that the laboratories *design* our cases, which amounts to *prescribing* for our patients. It is only natural for the laboratories to feel that they know more than we do about dentures, when, flocking to them for advice, we accept all the claims about the virtues of their dentures as gospel truth, and are carried away with the dazzling display of their wares, representing this man's or that man's secret process of construction.

Most partial dentures fail because of two reasons. They do not establish masticatory function; or it is accomplished (a) at the expense of the supporting teeth, which sooner or later are lost, or (b) at the expense of the supporting soft tissues and bone—the palate and the ridges—by inducing trauma and resorption. This condition invariably terminates in loss of masticatory function, thus completing a vicious circle.

In partial dentures we have to consider, *first*, the abutment teeth that are to be clasped. Broadly speaking, *at no time* should an abutment tooth be deprived of its freedom of movement in its socket as intended by nature. The abutment teeth should act only as stabilizers for the dentures, and, in turn, the clasps as supporters to the teeth without submitting them to violent intermittent pressure. In this way, loose teeth are tightened and are retained longer if properly supported during function. A state of normal function for clasped teeth cannot be brought about with cast clasps, half-round clasps, continuous cast clasps, or short round-wire clasps of a heavy gauge. Clasps made of the present available materials should never be heavier than 18 gauge. Gauges 19, 20, and 21 are the best for practically all partial cases. The greater the resiliency of a clasp, the less is the load on the tooth.

So much for clasps. Our next consideration is the supporting soft tissues, and the underlying bone. There is an old fundamental orthopedic law dealing with bone behavior under load and pressure. It is called Wolff's Law, and it reads as follows: "Every change in the form and position of the bones, or of their function, is followed by certain definite changes in their internal architecture, and equally definite secondary alterations of their external conformation, in

accordance with mathematical laws. To put the matter quite concisely, the external shape of the bone is the result of functional adaptation. The bone is strengthened and thickened at those points where most stress and pressure come upon it. And the adaptive transformation of the inner structure and outer shape of the bones takes place according to the altered demands." This law is being taken advantage of, and is applied, with remarkable results in both partial and full dentures. As we are dealing with injured tissues and injured bone, due to previous extraction, subjecting them to friction and load pressure, our problems of bone regeneration become fundamentally the same as the problems of an orthopedic surgeon in instances of similar nature.

We know that in all denture cases, the tissues that bear the brunt of the masticatory stresses are the mucosa and the ridges in lower dentures, and in addition to these, the palate, in upper dentures. Therefore, most of our denture cases fail because they are not properly based or properly fitted to the supporting tissues under load pressure. I prefer to call this proper fitting of the base of a denture to the tissues *the physiologic basing* of the denture. It means the construction of the base of a denture in such a manner that, when it is seated against the ridges and the palate under masticatory stress, the pressure over the whole surface of the supporting tissues is uniformly distributed, and so balanced that the pressure applied will be in direct proportion to the relative density of the underlying soft tissues, preventing the strangulation of blood vessels and the traumatization of underlying nerves. Dealing gently with these tissues will accomplish more than all the mechanical intricacies we can cramp into a denture. This idea is well understood by many prosthodontists who practice basing of dentures in a scientific manner. Why should we consider a denture completed when it is known that sooner or later destruction of tissue may ensue? Why dismiss a patient with the promise of future rebasing of his dentures when the mouth shrinks, when, by proper physiologic basing before the denture is delivered into the possession of the patient, rebasing in the future can be indefinitely postponed?

To make the idea perfectly clear, let us consider the sort of base obtained by means of the type of impression most generally used, namely an impression in plaster of paris. Plaster of paris gives a

faithful registration of the hard tissues of the mouth, that is, the teeth and their relation to one another, in a neutral state, but that is all. A cast made from a plaster impression is an accurate reproduction of actual conditions, so far as the teeth are concerned, but *not* of the soft tissues. In the first place, there is bound to be more or less distortion of these tissues during the taking of the impression, because the mouth is open, and because of the effect of the retrusion of the tongue upon its muscle attachments and the tissues of the floor of the mouth. There is a second reason, however, that is much more important. Plasters are soft, and, in fact, almost fluid before setting, and therefore they do not compress the mucosa. Consequently, they merely register the form of the soft tissue covering of the ridges, when not subject to pressure. This is very far from being the same as the form of the ridge under masticatory pressure, because the thickness, the density, and therefore the yield to pressure of the mucosa, are far from uniform. Therefore, a denture based upon a cast representing the condition of the tissues at rest, or uncompressed, would not fit those tissues in the same way when subjected to masticatory stress.

The dentures, whether made of gold or vulcanite, are hard, and during the masticatory process they are forced against the supporting ridges. The pressure applied to the denture by the opposing teeth, if well occluded, is equal over the whole area of the base of the denture, but the texture of the tissue overlying the bone is not such as to offer an equal resistance over the whole of the denture area. There are, so to speak, smaller areas or islands of less yielding tissue that receive all the masticatory stress, together with areas of thicker mucosa that receive no pressure at all. The inevitable result is trauma and resorption of the alveolar process. If bone alone were to suffer, or if the resorption were uniform, it would not be so disastrous. After the initial resorption of the thinly covered and more prominent bone areas, there might be an area of hard tissue covered with a uniform layer of mucosa, and further resorption might be arrested. This does not occur, however. The covering of the mucosa never becomes uniform in thickness, and it is not the bone alone that is affected. For, as the resorption proceeds, negative occlusion develops, the teeth are tilted and loosened. If they do not carry occlusal rests, the clasps ultimately become embedded in the cervical tissues, causing periodontal irritation, with consequent loss of the teeth.

The inadequacy of plaster impressions for the proper basing of partial dentures has been realized, and sectional impressions of modelling compound have been advocated. It is argued that as the denture must function under stress, therefore, an impression taken under stress or pressure will result in the production of a cast that is true to stress conditions. I fail to see, however, how hand pressure, applied to modelling compound harder than tissue, can produce the desired results. What is to prevent the depression of loose but serviceable teeth in their sockets, or their movement in the direction in which the hand pressure is applied? Also, would not the pressure applied to the lingual tissues adjacent to the anterior teeth of a lower case, for which a lingual bar is to be supplied, compress those tissues to such an extent as to make the placement of the bar at the proper distance from the lingual soft tissue area rather an uncertain matter? Apart from these objections, however, how can hand pressure upon the modelling compound be accurately gauged and directed so as not to be excessive, and yet to simulate the function pressure of mastication itself? It is most important to avoid excessive pressure, through too great tissue compression. These areas of bone and soft tissue have suffered injury through the extraction of the teeth. The healed over and filled in portion of the alveolar ridge is scar bone, and the overlying mucosa is scar tissue. Therefore, they are not normal. Being less vascular, as is all scar tissue, they are more susceptible to trauma, irritation, and resorption than uninjured tissues.

Then there is also another consideration. During the vertical movements of the mandible, the lower ridges are subjected to downward pressure, and during the lateral masticatory movements the underlying bone is subject to friction. Now, the normal behavior of tissues under load pressure, and friction, is too well known to be ignored. They react in direct proportion and in accordance to the nature of the external stimuli which they receive. It is this normal physiologic reaction of the tissues to pressure and friction that enables us to understand so clearly the difference between trauma and stimulation of the tissues, or better, the maintenance of the tissues in health, which is nothing but a *balance between waste and upkeep*. It has been pointed out that the ridge portions of the cast are merely superficial registrations of the form of the soft tissues, when not subject to pres-

sure. This is very far from being the same as the form of the ridges under masticatory pressure, not merely because the ridges are covered by a compressible layer of soft tissue, but because of the variation in the thickness and the compressibility of this tissue. It requires but a brief examination to discover areas like "islands" of hard, thinly covered bone amidst areas covered with thicker cushions of more compressible tissue.

Our principal reason, therefore, for the physiologic basing of these dentures, is to compensate for these tissue variations in a definite manner, so that when the dentures are seated against the ridges, under masticatory stress, the pressure upon each part of the surface will be relative to the compressibility of the tissue at that part. Consequently, everything depends upon the character of the impression material that is used and the distribution of the pressure upon the tissues while the impression is being taken. Theoretically, the material employed should have a similar degree of compressibility to the mucosa itself; should become plastic at as low a temperature as possible; and should be firm enough, when it has been cooled, to be handled with perfect safety when being flasked. In partial cases the physiologic basing is done after the denture is vulcanized and otherwise completed. A portion of the vulcanite, from its ridge surface, is removed and replaced with black-boxing wax, with which a reproduction of the tissues under actual masticatory pressure is obtained. The technic of this procedure is too well known to warrant my taking space to describe it.

III. FULL DENTURE CONSIDERATIONS

The physiologic basing requirements for partial dentures hold equally true for full dentures. Only, instead of completing the dentures and then basing them, we obtain the required results with a proper impression, which in technic is quite different from the commonly accepted compound or compound-and-plaster-wash technic. We are using less straight compound, or compound and plaster, impressions because of the overcompression of tissues which these materials will effect. Paradoxical as it may seem, a good plaster wash on a compound impression will compress the tissues more than if compound alone were used. It is the hydraulic pressure created with the fluid

plaster, when confined in the recesses prepared for it in the compound, that is excessive, plaster-escape provisions notwithstanding. With a plaster wash we defeat our own purpose. Instead of relieving pressure, as we intend to do, we create pressure.

The method described below provides for the impression to be taken under masticatory function, that is, the force is applied to the tissues by the patient's own masticatory effort and from a proper bite level, not by hand. The technic in brief is as follows: A good compound impression of the upper and lower jaws is taken, as for a plaster wash. Then a compound rim, built on both impressions to the proper bite length, is trimmed at the occlusal portion of the rim so that the patient is enabled to move his mandible laterally and forward, with easy sliding movements, without dislodging or tripping either of the impressions from its seat. By examining the mouth all the areas that need to be relieved are scraped from the compound impression, and both impressions are painted or coated with melted paraffin until the whole ridge area of the compound impression is covered with an even thin layer of paraffin. Both impressions are then inserted in the patient's mouth, and the patient is instructed to go through all the forward and lateral sliding masticatory movements possible for at least ten minutes. The impressions are then cooled with water and removed, and, if necessary, the operation is repeated until a good impression is obtained. No valve seal impressions can be obtained with this method, which is exactly what we wish to avoid. At mouth temperature, the paraffin yields enough, during the five or ten minutes that it is subjected to masticatory pressure, to adapt itself to the state of the tissues under functional pressure without over-compressing any particular area, neither strangulating the blood and nerve supply of the underlying tissues, nor traumatizing the bone of the palate and the processes.

Dentures constructed from such impressions are, from the start, tolerated better than any made by any other method. And although they do not exhibit any too great "suction" at first, they gradually develop one as the dentures are worn and function. The use of paraffin is not new in dentistry, nor is this technic wholly original. It has been modified to conform with our conception of function and maintenance of tissue health.

IV. ARTICULATION AND OCCLUSION

The next and most important consideration, for both partial and full dentures, is the establishment of functional occlusion. The best impressions cannot give us the results we are aiming at in dentures, if they are not made to function from the standpoint of occlusion. Balanced occlusion in dentures is a mechanical necessity. Without it, health in the oral cavity cannot be maintained. Without an efficiently balanced occlusion, nothing can stop the loosening of good healthy teeth, nor the destruction and resorption of the bone and the soft tissues of the mouth. We now have means by which we can obtain the necessary balance in dentures, without the elaborate theoretical details about the so-called "anatomical articulation" that in the past have been so confusing to most of us.

We do not attempt to restore to a patient sixty years old the occlusion of a person in the thirties. Neither do we aim to mutilate the occlusion of a young patient to that of an old one. We have ample means by which to *diagnose* our cases before attempting to restore them to function. In denture prosthesis we should never become so preoccupied with the mechanics of the undertaking as to forget how serious it really is because we are dealing with delicate living tissues. We should consider their tolerance for the appliance, and their response to the stresses or loads to which they are exposed through it. We must always bear in mind that the appliances are artificial, that the conditions under which these tissues were evolved by nature did not include the loads imposed by artificial dental restorations. Patients expect a certain degree of comfort, they expect a certain service from these appliances, and their expectations cannot be realized by the application of mere mechanics. There must be a thorough knowledge of the health requirements of each case, as well as a thorough knowledge of how to meet these requirements mechanically, either through our own efforts, or with the aid of a well trained technician or laboratory.

When anything considered fundamental cannot be disproved as such, and in due course it is called upon to serve as a specific measure, it should need no further individual interpretation as to whether it is wise or expedient to adopt it as a whole or modify it. As our conception

of dentistry as a profession becomes rooted more and more in fundamentals, the effects of the periodic epidemics of fanciful interpretations of phenomena that call for this or that remedy or technic will be felt less and less. In prosthetic dentistry the danger has been and still lies in the lack of adequate and thorough training to blend or coordinate our mechanical skill, from the standpoint of function and health, with the demands for it.

The better and more thorough our mechanical training, the less mechanical our dentures will be. A few months of intensive training in the simple fundamentals of the mechanics needed in dentistry would be ample foundation for all the dental students in our dental colleges. There is not a single mechanical operation in prosthetic dentistry that approaches or can compare with the wonders of the mechanical world. And yet for the lack of adequate mechanical training our progress in rendering better service to the public is much slower than conditions warrant. The fragmentary fashion in which mechanical laboratory technic courses are scattered through the first three years of the curriculum of most of the dental colleges accounts for a good deal of the uncertainty with which we handle our prosthetic cases.

When preventive dentistry becomes a fact, and teeth are lost no more, and pyorrhea goes the way of halitosis, then and only then will the demand for prosthetic services end.

483 Beacon Street,
Boston, Massachusetts.

TRIGEMINAL NEURALGIA[1]

FOSTER KENNEDY, M.D.
New York City

Trigeminal neuralgia is a disease in which, in nearly all cases, the dentist sees the patient before the physician does. The patient presents himself with the statement that he has neuralgia, pain in the teeth and face, and the dentist is asked to do, what is supposed to be, the needful extraction of teeth. I believe that the dentist usually fulfills this requirement because most of our trigeminal neuralgic patients present themselves to us minus most of their teeth! If dentists had a good understanding of this disease from the diagnostic standpoint, from the conception of pathology (highly speculative as it is), I think they would be slow to embark on an errand that must be without result.

Trigeminal neuralgia is preeminently a disease of middle life. Any person under thirty-five years of age complaining of neuralgia ought not to be diagnosed as such until the case has been thoroughly investigated. When I said thirty-five, I was exceedingly conservative. If I had said forty-five, I would still be within the general realm of truth. It is a disease preeminently of the age from forty-five to fifty-five. Many patients of course are sixty-five or seventy, but they usually have ten or fifteen years' history of pain. Persons with this disease are nearly all arteriosclerotic. It is a rare thing to find trigeminal neuralgia of the true type in a fat person with good blood vessels. They are not likely to have trigeminal neuralgia. But this terrible disease is usually found in thin people with degenerated blood vessels, not necessarily with high blood pressure but the kind of vascular degeneration seen easily enough with the ophthalmoscope, and in which the retinal arteries are observed to be tortuous and to reflect light as though from silver wire. Furthermore, if a patient presents

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himself or herself with the statement that pain is bilateral, sometimes felt in one side of the face and sometimes in the other and sometimes in both at once, you know almost certainly (although there is no rule in medicine without its exception) it is not a case of trigeminal neuralgia. If the patient says the pain is present for long periods of time, lasting fifteen or twenty minutes, several hours, or even more, you know he does not have trigeminal neuralgia, which is essentially paroxysmal.

The next diagnostic feature which you should scrutinize carefully is this: can the patient shave easily, provided the pain is in the second or third branch of the fifth nerve, or can he wash his face easily, because this pain is *excitable* to a preeminent degree. There is nearly always a "trigger zone" which, if irritated, will start off the attack of pain. When you ask such a person where the pain is, and he puts his hand boldly on the face, saying "It is here, Doctor," it is very unlikely he has trigeminal neuralgia! If he goes very delicately, like Agag, and is afraid to touch the skin of the face, you know that probably it is trigeminal neuralgia.

You have, therefore, a series of things that ought to help you: age, unilaterality, paroxysmal character with intervals, no remission but complete intermission of pain, and a trigger zone. These are four cardinal rules which I think you should always apply to any suspected case of trigeminal neuralgia. Any case of pain in the face ought to be asked these four questions. Then the attack itself. Anybody who has seen an attack of trigeminal neuralgia will always be able to identify another. The attack comes on often with a sharp twinge, not too bad at first, but terminating with a lightning-like, red-hot-wire-like pain in the face, which usually produces a spasm of the face partly as an expression of intense anguish. It is a pain not to be borne, one of the greatest a person is called upon to endure. It is practically self-limiting as regards the paroxysm. It lasts just a few minutes, but I once saw an attack of pain last twenty minutes. I never want to see another person experience anything like that. Only on this one occasion have I seen an attack of trigeminal neuralgia last for that period of time. Usually it lasts only fifty or sixty seconds, perhaps two or three minutes at most. The face is suffused. When the pain dies away the patient sinks back relaxed and exhausted by the effort of fighting such agony.

Usually when the second or third branch of the trigeminal nerve is involved, pain is experienced along that side of the tongue. That is an important anatomical consideration in diagnosis. If a person, allegedly having trigeminal neuralgia, tells you that the pain begins in the forehead and extends to the back of the head, over the head and to the back of the head and down the neck, be very careful of making the diagnosis of trigeminal neuralgia. If the pain goes outside the domain of the fifth nerve, it is probably not trigeminal neuralgia. It is remarkable how little overflow of pain there is in this disease, considering the terrific agony produced by it.

When young people have trigeminal neuralgia and the attack is unmistakable to your eye, then, in the light of the considerations that we have just made, you may suspect lues, diabetes, or tumor of the base involving the area in which the gasserian ganglion lies. There is usually some other gross factor of disease responsible for the production of the trigeminal picture. Lues and diabetes cause bilateral trigeminal neuralgia, but not often (lues very rarely). Remember, you do not have to find sugar in the urine to have diabetic pain. The worst form of degeneration of the posterior root ganglia is found in individuals in whom sugar does not spill over into the urine, but in whom there is a very high blood sugar. Such individuals may have very severe pain all over the body, what is known as diabetic neuritis, but which is really diabetic ganglionitis, affecting the posterior spinal root ganglia, and giving rise to changes in the gasserian ganglia. Such constitutional intoxications as that, metabolic intoxications, usually produce bilateral symptoms, so that true trigeminal neuralgia when bilateral ought to make you suspect diabetes, and not only diabetes but occult diabetes, i.e., diabetes in which the urine is normal but the blood chemistry is abnormal.

If you find that an individual having severe paroxysmal pain has at the same time anesthesia, a lowering of sensation in the affected skin area, you will do well to suspect tumor of the fifth nerve itself. That is by no means an invariable rule. In the last few months there came under our observation a distinguished man who had such an anesthesia and in whom the diagnosis of tumor was made on the basis of that anesthesia. It was found later no tumor existed. It is possible for functional trigeminal neuralgia to produce irritation of the

ganglia as well as some anesthesia of the face, so that one almost gets a condition of anesthesia dolorosa. (The word "functional" in this relation is complete assininity, for if ever there was an organic disease it is trigeminal neuralgia, but it is called functional disease because the ganglia have not been shown to be diseased.)

Then there is such a thing as *nuclear* pain. When we talk of trigeminal neuralgia, of course we mean neuralgia that is the result of a lesion, demonstrable or not as may be the case, of the peripheral apparatus of the fifth nerve. You will remember that the sensory root of the fifth is a very extraordinary structure. It is a long inverted cone extending through the bulb, bilaterally, to the upper part of the spinal cord. It has a very long stretch that can be diseased; and oddly enough, in spite of its being the nucleus of the nerve, pain can occur through its disease. I tried to distinguish for myself a little while ago the difference between nuclear pain and pain produced by irritation of the ganglion and the root. That of the ganglion and the root is, as has been described, paroxysmal, intermittent, lightning-like, agonizing, unilateral. That of the nucleus is usually bilateral by reason of the proximity of the two nuclei. Any disease of that area is apt to involve both nuclei, not necessarily but probably. The diseased process is continuous. It produces not the agonizing pain of trigeminal neuralgia of the peripheral type, but a sensation quite different. I asked a man in our service at Bellevue, who had such a lesion, to describe its effects, or rather I was telling a student that I thought the effect on the individual of such a lesion was the sensation of an exceedingly intense sunburn, and was gratified that the patient jumped from his seat in great excitement to verify that description. It seems to be a constant feeling of "glow" which almost becomes pain but is not at all an agonizing feeling. That is apparently the difference between the nuclear and the peripheral pain, a feeling of burning heat. "Glowing" perhaps is too benign a word by which to describe something which almost approaches pain. In this condition, the teeth have a feeling of being on edge.

I had a patient in Bellevue from one of the Veterans Bureaus. He had been an aviator who, having fallen some four hundred feet in a nose-dive, was badly broken up. Among various injuries he sustained was a fracture of the axis (the odontoid process), which should

have killed him, but did not. He had a temporary paralysis of both arms and both legs. I saw him five years later. He then had a feeling of constant pain in his teeth. That was his chief complaint—so much so, he could not work and he had been classified as a neurasthenic at the Veterans Bureau. I was put on the track of the possibilities of the case by its peculiar symptoms, and was able to find a metamere of the lowest part of the fifth nerve in which he could not feel at all well. There was an enormous depreciation of sensation in this skin area. What that fellow had suffered was really changes, possibly of a ganglionic character, in the nuclei of the fifth nerve, and he had the feeling of burning in his face and a constant pain in all of his teeth. As a result, I was convinced of the nature of his injury and I sincerely hope he was awarded a pension on that basis.

Sometimes one does not find pain or paroxysmal pain of the type occurring in trigeminal neuralgia, but there is complaint of tingling in the corner of the mouth and along the side of the tongue. That may be functional, but if it persists, it may mean a real involvement, by some organic lesion, of the fifth nerve. One of the most common of such organic lesions is tumor formation in the angle between the pons and the cerebellum. In that angle lie the fifth nerve supplying the face with sensation, the seventh nerve supplying motion to the face, and the eighth nerve supplying hearing. Rarely will you find involvement of the fifth coming under your particular notice without "ringing" in the ear of that side. It would be proper for the dental surgeon to inquire of such a patient whether there were noises like escaping steam or ringing on the side on which there is tingling of the edge of the tongue and the side of the face. Any weakness of movement of that side of the face would show that the seventh nerve also was involved and would lead to a fairly easy diagnosis.

What is the pathology of trigeminal neuralgia? Nobody knows. The gasserian ganglion has been sectioned in cases of trigeminal neuralgia, after having been removed as a curative measure, and at autopsy it was proved to be negative. Before I tell you what I think is the pathology of this disease, I will tell you of a patient who had this disease for some eight or ten years. At intervals of two, three, or four months at a time he would have no trouble, and then for periods of two, three, or four months he would have attacks once, twice, or a

dozen times a day, when he would have severe pain, always in the same side, always involving the second and third branch. I told him I believed the only proper treatment was an operation, but always advised patients to decide when that operation should occur. They knew their pain, and I tried to explain to them the risk of the operation, and the discomfort, such as they are. They make up their minds when the pain gets past the bearing point. I like my neuralgic patients to clamor for operation. It is better so. This man went away without any special treatment to think it over. He went hunting, on horseback, and was badly thrown. He received a violent blow on the chin and was unconscious for two or three minutes. He has never had the pain since. It is not a treatment I would always advise. I do not know how to apply the blow with enough skill to produce that result, but there was no doubt of the reality of his pain, I saw his paroxysms. There is no doubt of the diagnosis, and no doubt he has had no pain for the past three years. He had pain for eight or nine years before that. I believe a sclerosis of the petrosal plexus of vessels around the ganglion compresses and irritates the ganglion to the point of pain. I think this patient had that contact shaken loose by the blow.

As regards treatment: one can fatten these patients; that often helps. Give them coal-tar products, steer them clear of morphine. That is not so difficult because the pain is not continuous and they know it is self-limiting and will pass, but on the whole the medical treatment of real trigeminal neuralgia is very unsatisfactory. The disease usually progresses, gets worse, and the individual comes to operation. Schlosser introduced the method of injecting 80 per cent alcohol into the branches of the nerve, particularly the second and third branches. The injection of the first branch is a risky procedure because of the propinquity of the optic nerve. I have seen primary optic atrophy produced by injected alcohol that gained access to the optic nerve. I no longer advise injection of alcohol, because our neurological surgeons have become so skillful that the radical procedure (not removing the ganglion as used to be done, but of cutting the posterior root) is the operation of choice. Furthermore, if the pain is only felt in the third or second branch it is possible to dissect out only the fibres going to the second and third branches and cut them.

retaining and not cutting the fibres which go to the upper branch for the supply of the eye. Several of my patients had, as a residium of their operation, only an anesthesia of the third branch, the lower part of the jaw, a minor inconvenience to put up with in exchange for relief of the pain. Fifteen years ago my old chief, Sir Victor Horsley, would certainly have taken out the whole of the ganglion and produced complete anesthesia of the side of the face, but the methods of surgical treatment have been enormously refined. If I myself had trigeminal neuralgia, I would stand it until sure of the diagnosis and then have my root cut. I would not have an alcohol injection. That is surgery in the dark. It is very difficult to know where every drop of the 80 per cent alcohol is going.

I have various notes here regarding diseases of the bulb, either of ganglionic character producing anesthesia of the face, or dissociated change in facial sensibility sometimes with facial pain; also tabes—locomotor ataxia—which occasionally produces a curious mask of anesthesia with an underlying pain. But these matters are uncommon.

We neurologists have become enormously impressed with the manner in which infections reach the central nervous system. The central nervous system, enclosed in successive layers of membranes in a water jacket and all surrounded by a rigid bony box, seems to be as remote from external infections as anything could be. Furthermore, the connection between the spinal fluid and the blood system is blocked, the choroid plexus acting usually as a barrier to infection. It becomes a little difficult to understand how, except through sinus infections, the central nervous system becomes involved with toxemia. But we have been interested in finding out that the central nervous system can readily be reached by the perineural lymphatics. They are concomitant with and accompany the spinal roots, the cranial nerves. For instance, there are on record cases of mumps in which there is a progressive degeneration of the cells of the medulla occurring as a fatal complication. Diphtheria of the throat produces, of course, paralysis of the palate, not by inducing neuritis (the nerve is usually intact) but by obliterating or interfering with the functions and anatomical structure of the nuclei, so that the diphtheria toxin travels up the lymphatics of the nerves and assails the cells from which these

nerves proceed. Therefore it is certain that degeneration of the cells of the cord, and of the bulb of the brain, can be produced by toxins travelling up the perineural lymph paths. Undoubtedly pyorrhea, and chronic sepsis around the dental apices, may produce degeneration of cells in the central nervous system and in the cervical spinal cord. I leave the rest to you.

DISCUSSION

Charles A. Elsberg, M.D. (New York City): It has been my pleasure, during quite a number of years, to listen to papers read and addresses delivered at medical and dental societies and other associations, and also my misfortune (and that of the audience) to be asked to speak or discuss the papers presented. I divide the papers I have heard into two classes: One is the type of paper that tells a good deal—stimulates one's thought so that, when one speaks, one really has something to say. The second type, as represented this evening by Dr. Kennedy's address, covers the subject so completely, that not only is there nothing left to say, but if at one portion of the paper one was stimulated by some statement, the reader of the paper himself was sure to elaborate the remark a few minutes later.

That is the position I am in this evening, after having heard Dr. Kennedy's interesting remarks on trigeminal neuralgia and the facial pain of that affection. I say "affection" because I do not believe that we are justified, in spite of Dr. Kennedy's view, to speak of it as a "disease." The one very peculiar characteristic of trigeminal neuralgia, according to one neurologist, is that the attacks of pain, as described by Dr. Kennedy, occur only in the day time in from 50 to 70 per cent of the cases. When the patients go to sleep at night, they are free from pain until the following morning. This phenomenon is difficult to understand. If you have, for example, a toothache when you go to bed, you are unable to sleep because the pain continues. Therefore, it is hard to understand an affection in which the patient has pain which is so severe that, if it involves the second and third branches, it prevents him from chewing or swallowing food and he emaciates rapidly as the result; yet in many instances, as soon as he goes to sleep the pain is gone. I have wondered much about this, and I have experimented with a number of our patients from this viewpoint. I have tried to make them lie down in the day time, in the position they would occupy at night, and have found that in the day time they could not go to sleep and that their pain persisted. At night, they would go to bed at six or half past six or seven, fall asleep, and sleep until morning. That is an incomprehensible characteristic of the affection we call trigeminal neuralgia.

I always feel rather embarrassed, as regards my own self-respect, when a patient describes the pain he has had in his face, in the manner illustrated to you by Dr. Kennedy, and then, after speaking to him a few minutes, I am forced to tell him, "Yes, you have neuralgia." That simply means, "You have a pain in the face," which satisfies the patient and the members of his family who may be with him. We really know no more about it than the patient who has the pain. Therefore, the justification of the viewpoint expressed by Dr. Kennedy, that the patient who has the pain is the one to determine whether he desires to be subjected to a surgical procedure, which though practically non-dangerous, is and still must be called a major operation. In order to reach the fifth nerve one has to make an opening in the skull—one has to elevate the brain covered by the dura in order to reach the ganglion and the sensory root behind the ganglion.

The more I see of trigeminal neuralgia, the more I see of patients who, not as Dr. Kennedy has said, have had many of their teeth removed (and in this respect I cannot agree with Dr. Kennedy), but who have had *all* of their teeth removed in an effort to relieve them of pain. I really believe that the affection called trigeminal neuralgia has nothing at all to do with the teeth. Although extraction of teeth is the only method, by a lesser procedure, that physicians and dentists have to stop the pain, I believe the time will come when it will be regarded as a gross error to remove the teeth from a patient who has the characteristic symptoms of trigeminal neuralgia.

Functional disturbances (and one could recount many stories of patients with functional disturbances involving sensation in the face and giving the patient subjective pain) are sometimes greatly influenced by the removal of teeth. I well remember a lady, an intense neurotic, who every time before one of three children was born to her had intense pain in her face. I saw her before the first child was born. I said I did not believe she had trigeminal neuralgia. She went to a dentist, who removed several of her teeth in the area where she had pain, and instantly the pain stopped. Then she came back to me, and told me that it must have been neuralgia because several of her teeth had been removed and the pain had stopped. A year or two later, before the birth of the second child, the same thing occurred, and again she went to the dentist, and again several teeth were removed with the same result. Then, when she was about to have her third child, she again had pain and all of it on one side of the face. She did not come to me this time but went directly to the dentist (I did not blame her for that), and he removed the two remaining teeth. This time, however, her pain did not stop. Finally, at the end of a period of about four years she began to develop sensory disturbances in the face, and returned to me with all the symptoms of a tumor involving the gasserian ganglion.

Functional disturbances are sometimes difficult to distinguish from the real symptoms of neuralgia of the face, especially in neurotic individuals of the female sex. Because of the great severity of the pain, one is put to a great amount of trouble, and has to be exceedingly careful before subjecting a patient to an operation for supposed trigeminal neuralgia, when purely functional disturbances have not been fully excluded. I saw a patient who was slapped violently on the back by an intoxicated husband (in pre-prohibition days), and the result was the same as in the patient Dr. Kennedy told you of, who fell from his horse. I was more impressed by the fact that this patient had a functional disturbance, although the explanation given by Dr. Kennedy is an exceedingly interesting one.

Aside from the conditions mentioned by Dr. Kennedy (of course in his short remarks he could not mention all of them), there is a very interesting disturbance that occurs in the muscles of the face, especially in the masseter and sometimes in the internal pterygoid, with which pain is associated in the face and along the gums, and which promptly yields to mechanical therapeutic measures. I have seen a patient who, having what appeared to be a typical toothache, coming on day after day, had his infected teeth removed, but was not benefited, yet was relieved by thorough massage and light treatment of the muscles of the face. Myositis can cause symptoms very similar to those of a second or third branch neuralgia. Then there is a type of neuralgia, exceedingly interesting, which we have only begun to recognize during the last few years. This type has been recognized because these patients were supposed to be suffering from trigeminal neuralgia. Although the sensory root was divided, the pain still continued, having been brought on by chewing or swallowing food, and usually was located in the most posterior part of the lower jaw or the adjoining portion of the tongue, at the root of the tongue, and the soft palate. Lately, it has been recognized that there is a group of patients with neuralgia of the glossopharyngeal nerve, which is very difficult to distinguish from neuralgia of the trigeminal nerve. Finally, as Dr. Kennedy mentioned, a patient may have a type of pain which, beginning in the face and extending over the head, may reach into the neck, to the shoulders, or to the arm, yet is not trigeminal neuralgia. A patient with trigeminal neuralgia has his pain localized over the area of distribution of the first, second, or third, or of two or of all of these branches of the fifth nerve.

Because of the fact that we do not know what this affection is, what the substratum is upon which it is based, why the attacks occur in such a particular way, brought on sometimes only through exposure to cold, as some have their attacks of neuralgia in the winter time—others are brought on by

the warm weather—I believe one should be as conservative as possible before subjecting the patient to a major surgical procedure. There is a considerable group of patients who can be relieved without surgery by properly given alcohol injections. There are a certain number of persons that can receive an alcoholic injection every one, two, or three years, and then be saved from operative procedure for the rest of their lives. I have had several old ladies and gentlemen who have had repeated alcohol injections, which have kept the patients well for the remainder of their lives. The same has been true of patients with high blood pressure, with blood pressure of 200 to 250, whose chances of life are relatively poor because they are apt to have a rupture of a cerebral vessel, and apoplexy sooner or later from their high blood pressure and arterial changes. These patients very often can be kept perfectly happy and comfortable for their remaining years by alcohol injections. Personally, I do not like to give alcohol injections. As Dr. Kennedy mentioned, I believe one is working in the dark with this treatment. Whenever I can, I turn such a patient over to one of my assistants or associates. As Dr. Kennedy mentioned, an injection into the first branch should rarely be made on account of the danger that alcohol, entering the orbit will cause edema and loss of the eye; or that alcohol, reaching the optic nerve, will have an effect such as that in one of Dr. Kennedy's patients. Injections into the second and third branches can be given, but they should usually be deep, just beyond the ganglion. Peripheral injections into the infraorbital and mental foramina do little good, which lasts only a short time. So, alcohol injections must be deep in the second and third branches, just beyond their exit from the foramina of the skull.

I do not think it necessary to speak at length regarding the modern operation for trigeminal neuralgia. Great advances in method and technique have been made in the original work by Dr. Frazier and Dr. Spiller, at the University of Pennsylvania, and carried on in my clinic by one of my associates, Dr. Stookey. These advances have developed the operation for trigeminal neuralgia to the perfect state in which it exists to-day.

Finally, regarding the entire subject of pain in the face, it must be said that at least one-half of the patients who consult me for supposed trigeminal neuralgia do not have it. They have either functional disturbances or new growths within the cranial cavity involving the fifth nerve—either new growths derived from the eighth nerve, as Dr. Kennedy mentioned, or new growths lying in the most anterior portion of the cranial cavity, in or under the frontal lobe of the brain. Therefore, the dentist, the neurologist, and

the surgeon must be certain the patient has true trigeminal neuralgia before subjecting him to treatment for it, whether the treatment be alcohol injections, or the more radical operation of division of the root or part of the sensory root by the surgical procedure that has been developed in recent years.

President Arthur H. Merritt: In arranging this important program, the Committee was of the opinion that the Fellows of the Academy would rather be listeners than discussers. However, if any one has anything to say or any question to ask, we should be glad to hear from him.

Edward G. Van Valey, D.D.S. (*New York City*): Does the extraction of teeth in true trigeminal neuralgia give any relief, and if so, is it only temporary?

William D. Tracy, D.D.S. (*New York City*): I am not qualified to discuss the subject before us this evening, but I would like our guests to know that I believe all the Fellows here feel this has been a very interesting and profitable evening for us. I presume there are very few who have not occasionally been troubled in trying to reach a decision in regard to obscure pains. We have read profoundly and listened to discourses, and I think we must be impressed by the thoroughness and scientific manner in which our guests have presented the subject. They profess ignorance of those things on which they have no light, and that shows their conservatism. Personally, I am grateful for their coming.

Dr. Kennedy (*in conclusion*): I do not think, Dr. Van Valey, extraction does any good at all. It is a condition where the trouble with the plumbing lies in the cistern, and taking out the teeth is like tinkering at the pipes. It has no value. It usually leads to a good deal of mental distress through blighted hopes.

Dr. Elsberg was very wise in speaking of the odd characteristic of trigeminal neuralgia by which it appears in the day time and not during sleep at night. We do not know much about the condition of the vascular apparatus within the skull in sleep. We do not yet know what sleep is, though we spend a third of our lives in it. I think the fact that trigeminal neuralgia does not appear in sleep is a very potent argument for the view that it has a vascular basis, as I suggested.

I agree that alcohol injections should be used where the operation of the

opening of the skull is contra-indicated, by reason of age, or of advanced arteriosclerosis. I believe it is a major operation as regards shock. It is not a major operation as regards procedure. It is very painful. The patients that I have injected have told me that the pain is worse than their paroxysms. One can well believe it. I think it is a matter to avoid. The better thing to suggest is the operation Dr. Elsberg knows so well how to do.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH[1]

A Brief Outline of Its Organization and Early Development

WILLIAM J. GIES, F.A.C.D.

Editorial Office, Journal of Dental Research

During the earlier years of the writer's endeavors in the field of dental research, he was greatly impressed by the general indifference to original investigation in stomatology that then prevailed not only among dental and medical practitioners, but also among the teachers of biology, of medicine, and of dentistry. The need for effectual agencies for widely concerted promotion of research in dental science and art, analogous to those in medicine and the medical sciences, gradually became so obvious that in the spring of 1917 he inaugurated a definite plan for the more rapid advancement of dental research by three important conventional means; namely, a journal, an international association, and improvement of the status and of the support of research in the dental schools and universities.

During the progress of the preliminary effort to establish the *Journal of Dental Research* (1917–1919), which was the first of the three agencies to receive direct attention, an endeavor to found an international association was initiated. Believing that such an association could best be formed as a federation of local societies, each to be an autonomous section of a national division of the international organization, and confident that it could be projected effectually in the largest city in the United States, the writer corresponded or conferred orally with about one hundred of the leading dentists in New York

[1] Reprinted, with several additional footnotes, from the *Journal of Dental Research* (1926, vi, pp. 101–111), in order, for the convenience of all concerned, to assemble in the present volume, with the associated accounts of proceedings, a complete record of the organization and work of the Association for the years 1920–27, inclusive. See the record of scientific proceedings, etc., beginning on page 209 (this issue); and of the executive proceedings, in the supplement of this volume.

regarding the feasibility of the general plan and the personnel of the charter membership. Occasional private ballots during a period of about two years (1919–1920) having clearly crystallized opinion, the men who had been selected by this democratic procedure were invited to dine at the Columbia University Club, at 4 West 43rd Street, New York City, on the evening of December 10, 1920, to consider the advisability of promoting research by international organization. The names of those who attended the dinner are appended:

E. A. Bogue
Adolph Berger
Theodor Blum
George W. Clapp
Henry S. Dunning
William J. Gies
Henry W. Gillett
R. G. Hutchinson, Jr.
Thaddeus P. Hyatt
Milo Hellman
Victor H. Jackson
F. C. Kemple
Arthur H. Merritt
Bissell B. Palmer, Jr.
Frederick A. Peeso
M. L. Rhein
James P. Ruyl
Paul R. Stillman
Leuman M. Waugh
J. Leon Williams
J. Lowe Young

Drs. Herman E. S. Chayes, William B. Dunning, M. I. Schamberg, and Frank T. Van Woert were unavoidably prevented from being present, but had indicated complete accord with the tentative plan for the organization of the proposed association.

After the conference had been organized by the selection of J. Leon Williams as Chairman, and Leuman M. Waugh as Secretary, the writer formally outlined the steps that led to the meeting, and included a statement to the effect that dentists in Boston and Chicago, who had been consulted, were ready to endorse the general proposal and to hold meetings promptly for the organization of sections. An earnest approbative discussion eventuated in a unanimous vote to organize the proposed association by the adoption of the following Articles of Agreement, which previously had also been submitted to and informally approved by the earliest of the prospective members in Boston and Chicago.

ARTICLES OF AGREEMENT

EFFECTING THE ORGANIZATION AND PROVISIONAL GOVERNMENT OF THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Founded in New York City, December 10, 1920

PREAMBLE

In order to promote broadly the advancement of active research in all branches of dentistry, and in related phases of the arts and sciences that contribute directly to the development of dentistry; and, further, to encourage and facilitate coöperative effort and achievement by, and mutual helpfulness among, investigators in all nations in every division of stomatology—to the end that dentistry may render cumulatively more perfect service to humanity—we, the undersigned, assembled at the Columbia University Club, in New York City, December 10, 1920, hereby establish the International Association for Dental Research, and, as its founders, subscribe to and adopt these Articles of Agreement for the provisional government of this Association.

ARTICLE I. OFFICIALS

SECTION 1. *Provisional government.* The provisional executive management of this Association shall be vested in a Council of eleven members, at least six of whom shall be elected at this meeting of organization.

SECTION 2. *Officers.* The officers of the Council, to be elected by the Council itself, shall serve as the officers of the provisional government of this Association.

ARTICLE II. INSTRUCTIONS TO THE COUNCIL

SECTION 1. *Preparation of a constitution.* The Council to be elected is hereby instructed to prepare a constitution for our permanent government, to replace these Articles, and to submit to us such a constitution for formal consideration before February 1, 1921. The Council is also charged to consider ways and means for the permanent international establishment of this Association, and for the immediate progressive furtherance of the Association's purposes. The Secretary of the meeting at which these Articles are adopted shall transmit a copy of these Articles to each member of the Council to be elected.

SECTION 2. *Meetings of the Council.* The Council may, by a majority

vote of its total membership, call meetings of the Council after December 18, 1920; and, by such a vote, the Council may use any means at its discretion for prompt procedure toward attainment of the objects of this Association.

Article III. Meetings

Section 1. *Next meeting.* The next meeting of the Association shall be held at the Harvard University Club, on December 17, 1920, when three additional members of the Council may be elected.[2]

Section 2. Meetings of the Association may be called by the Council on its own initiative, and shall be called at the request of five members of the Association.

Article IV. Absent Colleagues Elected to Charter Membership

Having indicated direct interest in the successful foundation of this Association, but having been unavoidably prevented from attending this meeting of organization, those of our colleagues who are named below, in this Article, are hereby unanimously elected to membership as founders of this Association:

New York.—Herman E. S. Chayes, William B. Dunning, M. I. Schamberg, Frank T. Van Woert.

Boston.—Robert R. Andrews, Lawrence W. Baker, George A. Bates, Fred R. Blumenthal, Percy R. Howe, A. Leroy Johnson, V. H. Kazanjian, L. M. S. Miner, William Rice, Alfred P. Rogers, H. Carlton Smith, Eugene H. Smith, Kurt H. Thoma, George H. Wright.

Chicago.—Frederick B. Noyes.

Article V. Sections of the Association

The members in any geographical group may organize local sections of this Association; but they may not do so prior to the adoption of a Constitution that will prescribe the relationships of such sections to the Association in general.

Article VI. New Members

Pending the adoption of a Constitution to replace these Articles of Agreement, new members may be elected by a majority vote of the members

[2] At that meeting the members were authorized to hold a special meeting in Chicago, on December 30, 1920, to complete preliminaries of organization, including the election of two additional members of the Council.

in any geographical section, subject to confirmation by a majority vote of the total membership of the Council.

ARTICLE VII. QUORUM

Nine members shall constitute a quorum for the transaction of business by this Association until these Articles of Agreement for our *provisional* government will have been superseded by a Constitution, for our *permanent* government, adopted in conformity with these Articles.

(Signed by all named above as present at the meeting of organization.)
Attest:

J. LEON WILLIAMS, *Chairman.*

LEUMAN M. WAUGH, *Secretary.*

On December 17, 1920, Dr. J. Leon Williams and the writer attended the second meeting of the Association, at the Harvard Club in Boston, where the Boston members named in the foregoing Articles of Agreement laid the foundation for the Boston Section, and additional members of the Association, chiefly resident in Boston and Chicago, were elected. On December 30, 1920, at the University Club in Chicago, the writer, in conformity with action at the Boston meeting, presented the plan to the Chicago members and their guests, who then took the preliminary steps for the establishment of the Chicago Section, and elected additional members. The election of new members at the Boston and Chicago meetings was subsequently ratified by the Association.

In accordance with the requirements of the foregoing Articles of Agreement, the Council was organized promptly by representatives of the New York (6), Boston (3), and Chicago groups (2), and by the spring of 1921 the appended Constitution, prepared by the Council, had been adopted for the government of the Association.

[*General informal memorandum.*—The following Constitution created an international body composed of national (general) divisions containing intranational (local) sections. The divisions and sections are autonomous in accordance with the limitations of ordinary federation. The objects, as stated in Article II, include the primary purpose to provide meetings for the encouragement and assistance of those actually engaged in research. Members, who are elected by the Association at annual meetings only, may organize divisions in any nation and sections in any division. Anyone who has conducted and published an account of a meritorious original investigation in dental science or art, or in

any of the sciences contributory to stomatology, is eligible to membership. A Council, consisting of elected representatives of the present Sections, serves as an executive committee during the intervals between annual meetings, which are devoted primarily to the presentation and discussion of papers on research by the members and their guests. Article III prohibits commercial relationships between the Association and individuals or organizations. Bylaws have not yet been adopted.]

CONSTITUTION OF THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

ADOPTED IN CONFORMITY WITH THE PROVISIONS OF THE *Articles of Agreement* ON WHICH THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH WAS FOUNDED IN NEW YORK CITY ON DECEMBER 10, 1920, AND WHICH ARTICLES OF AGREEMENT THIS CONSTITUTION SUPERSEDES AND REPLACES

ARTICLE I. NAME

This organization is named: *International Association for Dental Research.*

ARTICLE II. OBJECT

The Association has been established (a) to promote broadly the advancement of active research in all branches of dental science and in related phases of the sciences that contribute directly to the development of dentistry, and which add to the knowledge of the mouth and teeth and of their relations to the body as a whole; and further, (b) to encourage and facilitate coöperative effort and achievement by, and mutual helpfulness among, investigators in all nations in every division of stomatology; (c) to the end, particularly, that dentistry may render cumulatively more perfect service to humanity.

ARTICLE III. PROHIBITION OF COMMERCIAL RELATIONSHIPS

This Association shall in no way enter into, or foster, relations with commercial organizations, nor with makers of appliances or of anything else used in the art of dentistry by the dental profession, or in the care and treatment of the oral structures by the laity.

ARTICLE IV. MEMBERSHIP

SECTION 1. *Eligibility.* Any person who has conducted, and published an account of, a meritorious original investigation in dental science, or in any of the sciences contributory to stomatology, shall be eligible to mem-

bership in this Association, providing such person conforms to the recognized standards of professional ethics.

SECTION 2. *Nomination.* (A) Nominations to membership may be made at any time by any member. Each nomination must be formally endorsed by two additional members, who know personally and certify from specific knowledge, that the candidate for membership is eligible, before the nomination can be registered for official presentation to the Association.

(B) Each endorsed nomination for membership must be formally presented to the Council of the Association, with three copies each of one or more publications which show that the nominee meets the requirement of eligibility relating to research achievement.

(C) The Council shall determine, in the case of each nomination, whether the nominee fully meets the requirements of eligibility. The Council shall report promptly to the Association the details of its findings in this relation, and also such recommendations as it may wish to offer with its report.

SECTION 3. *Election.* (A) New members may be elected only at annual meetings of the Association.

(B) Election to membership shall be by ballot by the *individual* members. A two-thirds affirmative vote of the ballots cast shall be necessary for election. Substitution of any other parliamentary procedure for that of a secret ballot shall invalidate all elections to membership that may be accomplished in such an irregular way.

SECTION 4. *Initiation.* Initiation of membership in this Association requires (a) formal approval of a certified copy of this Constitution, signified to the Secretary of the Council; also (b) the member-elect's formally avowed acceptance of, and adherence to, all the provisions of this Constitution and of the by-laws of the Association. No member-elect can exercise the privilege of membership before these requirements are met.

SECTION 5. *Forfeiture.* Membership in this Association may be terminated by a member at any time by formal notice, to the Secretary of the Council, of that member's withdrawal. Delivery of such a notice automatically effects that member's withdrawal from membership in this Association.

The privilege and rights of membership may be terminated by the Association for any member at any time, by formal notice to a member, from the Secretary of the Council, of the Association's decision, at an annual meeting by a two-thirds vote of those present and voting, that the member's association is no longer desired. Delivery of such a notice to a member automatically terminates his or her membership in this Association.

Annual dues for the fiscal year in which membership may be terminated by a member or by the Association shall be repaid or, if unpaid, shall not be demanded.

Article V. Divisions of the Association[3]

Section 1. *Organization.* The members in any nation, with the approval of the Association, may organize a Division of the Association, particularly for the national advancement of the objects of the Association.

Section 2. *Management.* The national affairs of a Division shall be managed by its own members, but such management must conform in all respects with the requirements of this Constitution and of the by-laws of the Association.

Article VI. Sections of the Association

Section 1. *Organization.* The members in any geographical center, with the approval of the corresponding national Division, may organize a Section of the Association, particularly for the local advancement of the objects of the Association.

Section 2. *Management.* The affairs of the Sections shall be managed by their own members, but such management must conform in all respects with the requirements of this Constitution, with the by-laws of the Association, and with the by-laws of the corresponding national Division of the Association.

Article VII. Meetings

Section 1. *Annual.* The Association shall meet at least once annually, beginning with the calendar year 1921, at times and places to be determined by the Association, or by the Council when it is authorized to act for the Association.

Section 2. *Special.* Special meetings of the Association may be convened by the Council, and shall be called at the request of one-fifth of the total membership.

Article VIII. Officials

Section 1. *Council.* (A) Authority. During the periods between annual meetings, the executive management of the affairs of the Association shall be conducted by the Council, within the general authority voted to it by the Association.

[3] Divisions have not yet been organized.

(B) Personnel. (a) The Council shall consist of three elected representatives of each national Division. Until the number of divisional representatives in the Council attains a total of at least fifteen, the number necessary to constitute that total shall be elected from the American Division of the Association.

(b) With the adoption of this Constitution, the terms of all the members of the existing Council of the Association (selected on the basis of the provisional organization) shall close automatically, when it shall become the duty of each of the three existing and ratifying Sections[4] of the Association to elect by ballot five Councillors, the fifteen Councillors thus elected to serve as, and to be, the Council of the Association, until their successors will have been chosen in accordance with the provisions of this Constitution.

(C) Election of the divisional representatives. The representatives of the national Divisions in the Council shall be elected by the respective national Divisions.

(D) Length of the term of service. The length of the term of service of the individual representatives in the Council shall be determined by the respective national Divisions.

SECTION 2. *Officers.* (A) Identical with those of the Council. The general officers of the Council shall be, also, the officers of the Association. They shall be nominated by the Council, preferably though not necessarily from its own membership, subject to formal confirmation by the Association.

(B) Nomination. The list of nominations for the offices of the Council and Association, for a given term, shall be formally announced by the Council to each member of the Association at least three months prior to the date of the annual meeting at which the same nominations will be presented to the Association for its consideration.

(C) Election. The Council's nominees for the offices in the Council and Association, if approved by a majority vote of those present and voting at the succeeding annual meeting, shall become the officers of the Council and of the Association. In the event, however, that the Association should record a majority vote against any of the Council's nominees for the general offices, the Council, at the same meeting, shall present one or more additional nominations for the corresponding offices, until the Association votes its approval. But should the Council fail to make satisfactory nominations, the Association may then and there, independently of the Council, complete the nomination and election of general officers of the Council and Association.

[4] This was a formal recognition of the existence of the New York, Boston, and Chicago Sections, which until then were informal "groups."

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bership in this Association, providing such person conforms to the recognized standards of professional ethics.

SECTION 2. *Nomination.* (A) Nominations to membership may be made at any time by any member. Each nomination must be formally endorsed by two additional members, who know personally and certify from specific knowledge, that the candidate for membership is eligible, before the nomination can be registered for official presentation to the Association.

(B) Each endorsed nomination for membership must be formally presented to the Council of the Association, with three copies each of one or more publications which show that the nominee meets the requirement of eligibility relating to research achievement.

(C) The Council shall determine, in the case of each nomination, whether the nominee fully meets the requirements of eligibility. The Council shall report promptly to the Association the details of its findings in this relation, and also such recommendations as it may wish to offer with its report.

SECTION 3. *Election.* (A) New members may be elected only at annual meetings of the Association.

(B) Election to membership shall be by ballot by the *individual* members. A two-thirds affirmative vote of the ballots cast shall be necessary for election. Substitution of any other parliamentary procedure for that of a secret ballot shall invalidate all elections to membership that may be accomplished in such an irregular way.

SECTION 4. *Initiation.* Initiation of membership in this Association requires (a) formal approval of a certified copy of this Constitution, signified to the Secretary of the Council; also (b) the member-elect's formally avowed acceptance of, and adherence to, all the provisions of this Constitution and of the by-laws of the Association. No member-elect can exercise the privilege of membership before these requirements are met.

SECTION 5. *Forfeiture.* Membership in this Association may be terminated by a member at any time by formal notice, to the Secretary of the Council, of that member's withdrawal. Delivery of such a notice automatically effects that member's withdrawal from membership in this Association.

The privilege and rights of membership may be terminated by the Association for any member at any time, by formal notice to a member, from the Secretary of the Council, of the Association's decision, at an annual meeting by a two-thirds vote of those present and voting, that the member's association is no longer desired. Delivery of such a notice to a member automatically terminates his or her membership in this Association.

Annual dues for the fiscal year in which membership may be terminated by a member or by the Association shall be repaid or, if unpaid, shall not be demanded.

Article V. Divisions of the Association[3]

Section 1. *Organization.* The members in any nation, with the approval of the Association, may organize a Division of the Association, particularly for the national advancement of the objects of the Association.

Section 2. *Management.* The national affairs of a Division shall be managed by its own members, but such management must conform in all respects with the requirements of this Constitution and of the by-laws of the Association.

Article VI. Sections of the Association

Section 1. *Organization.* The members in any geographical center, with the approval of the corresponding national Division, may organize a Section of the Association, particularly for the local advancement of the objects of the Association.

Section 2. *Management.* The affairs of the Sections shall be managed by their own members, but such management must conform in all respects with the requirements of this Constitution, with the by-laws of the Association, and with the by-laws of the corresponding national Division of the Association.

Article VII. Meetings

Section 1. *Annual.* The Association shall meet at least once annually, beginning with the calendar year 1921, at times and places to be determined by the Association, or by the Council when it is authorized to act for the Association.

Section 2. *Special.* Special meetings of the Association may be convened by the Council, and shall be called at the request of one-fifth of the total membership.

Article VIII. Officials

Section 1. *Council.* (A) Authority. During the periods between annual meetings, the executive management of the affairs of the Association shall be conducted by the Council, within the general authority voted to it by the Association.

[3] Divisions have not yet been organized.

(B) Personnel. (a) The Council shall consist of three elected representatives of each national Division. Until the number of divisional representatives in the Council attains a total of at least fifteen, the number necessary to constitute that total shall be elected from the American Division of the Association.

(b) With the adoption of this Constitution, the terms of all the members of the existing Council of the Association (selected on the basis of the provisional organization) shall close automatically, when it shall become the duty of each of the three existing and ratifying Sections[4] of the Association to elect by ballot five Councillors, the fifteen Councillors thus elected to serve as, and to be, the Council of the Association, until their successors will have been chosen in accordance with the provisions of this Constitution.

(C) Election of the divisional representatives. The representatives of the national Divisions in the Council shall be elected by the respective national Divisions.

(D) Length of the term of service. The length of the term of service of the individual representatives in the Council shall be determined by the respective national Divisions.

SECTION 2. *Officers.* (A) Identical with those of the Council. The general officers of the Council shall be, also, the officers of the Association. They shall be nominated by the Council, preferably though not necessarily from its own membership, subject to formal confirmation by the Association.

(B) Nomination. The list of nominations for the offices of the Council and Association, for a given term, shall be formally announced by the Council to each member of the Association at least three months prior to the date of the annual meeting at which the same nominations will be presented to the Association for its consideration.

(C) Election. The Council's nominees for the offices in the Council and Association, if approved by a majority vote of those present and voting at the succeeding annual meeting, shall become the officers of the Council and of the Association. In the event, however, that the Association should record a majority vote against any of the Council's nominees for the general offices, the Council, at the same meeting, shall present one or more additional nominations for the corresponding offices, until the Association votes its approval. But should the Council fail to make satisfactory nominations, the Association may then and there, independently of the Council, complete the nomination and election of general officers of the Council and Association.

[4] This was a formal recognition of the existence of the New York, Boston, and Chicago Sections, which until then were informal "groups."

(D) Elective term of service. The elective term of service in all the general offices of the Council and Association shall begin at the close of an annual meeting and shall terminate at the end of the next succeeding annual meeting.

Article IX. Finances

Section 1. *Amount of dues.* The annual dues, payable by each member, before the close of each annual meeting of the Association, shall *not exceed* five dollars ($5.00) or its approximate equivalent.

Section 2. *Payment of dues.* The annual dues may be paid through the agency of national Divisions.

Section 3. *Restriction on the expenditure of funds.* No funds of the Association may be expended by the Council without general authorization by, or approval of, the Association.

Article X. By-Laws

By-laws of this Association may be adopted at any meeting of the Association by a two-thirds vote of the members present and voting.

Article XI. Quorum

Fifteen members shall constitute a quorum of this Association.

Article XII. Amendments

Section 1. *Constitution.* (A) Presentation. Proposed amendments to this Constitution may be presented, at annual meetings of the Association, by and with the formal endorsement of five members. Statements of reasons for the adoption of proposed amendments must accompany the formal presentation of each such amendment.

(B) Notification of all the members. It shall be one of the duties of the Council to deliver to each member of the Association, within six months of the date of the annual meeting at which an amendment was proposed, (a) a printed copy of each such suggested amendment, with the names of the sponsors and the reasons given formally for its proposed adoption; also (b) a blank ballot for the registration of a vote for or against the proposed amendment.

(C) Adoption. At the next, or any, succeeding annual meeting, the foregoing conditions having been met in detail, a vote on the proposed amendment shall be in order; and the proposed amendment shall become a part of this Constitution if, and when, at least two-thirds of the entire membership of the Association vote for its adoption.

SECTION 2. *By-laws.* Amendments to the By-laws may be adopted at any meeting of the Association by a two-thirds vote of those present and voting.

[By-laws have not yet been adopted]

Ratified for the Association, by the New York, Boston, and Chicago Sections, on May 27, 1921.

Attest:

J. Leon Williams, *President.* Leuman M. Waugh, *Secretary.*

Since the adoption of the foregoing Constitution, which is still in force, sections of the Association have been organized in Toronto (1921), Ann Arbor (1923), and San Francisco (1924), and successful annual meetings have been held in New York, Chicago, and Washington, D. C.[5] Accounts of the past proceedings of the several sections and of the annual meetings of the Association will be included in early issues of the *Journal of Dental Research.*[6] The regular presentation in this *Journal* hereafter, of the scientific proceedings of the Association and of its Divisions and Sections, promises to further the cumulative general development of dental research as effectually as the publication of the similar *Proceedings of the Society for Experimental Biology and Medicine* has activated medical research in North America since 1904.

The Association was organized to promote research, nothing else; and is wholly impersonal in character and avowedly non-political in purpose.[7] Its development has been slow but spontaneous; and its activities, however restricted, have beeen natural not artificial. It has not been forced to walk before it was able to stand. Although the Association has not been much stronger than the slowly developing spirit of research in the dental profession, and has shown unmistakable symptoms of recurrent rickets, nevertheless the food which dental research is now receiving is better balanced and more nutritious than it has been. A further improvement of the present diet, which

[5] The elections to membership at the Sixth Annual Meeting of the Association, held in Washington, D. C., March 25–28, 1928, indicate that additional sections are about to be organized in Halifax, Minneapolis, Philadelphia, Pittsburgh, and St. Louis.

[6] See the *Journal of Dental Research*, 1928, viii, pp. 7 and 209.

[7] Williams: *Journal of Dental Research*, 1923, v, p. 27.

contains increasing proportions of the vitamins of financial sustenance, will cause more rapid growth of dental research, and will also assure the Association's attainment of health, vigor, activity, and fecundity. It is hoped that additional sections will be organized in this country and Canada, and abroad.

The first published reference to the International Association for Dental Research was appended to an appreciation of Robert R. Andrews by George A. Bates,[8] both of whom, as founders of the Association, attended the meeting for the organization of the Boston Section, and spoke earnestly in behalf of international accord for the advancement of dental research. Andrews and Bates, distinguished for high character as men and dentists and teachers, exemplified, in their devotion to idealism and scholarship and truth, the spirit and the abiding aspirations of the International Association for Dental Research.

[8] Editorial comment: *Journal of Dental Research*, 1921, iii, p. 5 (footnote).

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

A Summary of the Proceedings of the General Meetings Since Organization, in New York City, in 1920[1]

Compiled by WILLIAM J. GIES, F.A.C.D.[2]

Editorial office, Journal of Dental Research

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A. SCHEDULE OF GENERAL MEETINGS

a. Organization[3]

(a) Establishment: New York, December 10, 1920.[3]

(b) Further organization: Boston, December 17, 1920.[3]

(c) Further organization: Chicago, December 30, 1920.[3]

(No section was officially organized before the adoption of the Constitution on May 27, 1921. The New York, Boston, and Chicago Sections were organized *informally*

[1] A brief outline of the organization and early development of the Association was published in the *Journal of Dental Research*: 1926, vi, p. 101. To serve the convenience of all concerned, a coördinated record of the proceedings of the Association since its organization will be published in the present volume. For this reason the outline is reprinted, with additions, in this number: pp. 197–208. Hereafter the proceedings of the Association and Sections will be published promptly in this *Journal*.

[2] Most of the abstracts have been prepared by the authors of the corresponding papers. All have been verified by them.

[3] See the reprinted outline of these meetings on pages 198–207, and the explanation in footnote 1.

before that date; the Toronto Section was then being created; the Ann Arbor and San Francisco Sections were established in 1923 and 1924, respectively.

b. Scientific sessions[4]

None in 1920 or 1921.[4]

First: New York, December 21, 1922. Guest of the New York Section.

None in 1923.[4]

Second: Chicago, March 7, 1924. Guest of the Chicago Section.

Third: Chicago, March 20, 1925. Guest of the Chicago Section.

Fourth: Chicago, March 26, 1926. Guest of the Chicago Section.

Fifth: New York, April 19, 1927. Guest of the New York Section.

B. SCIENTIFIC PROCEEDINGS: ABSTRACTS OF THE PAPERS PRESENTED AT THE ANNUAL MEETINGS

I. First Meeting: December 21, 1922, Columbia University Club, New York City

Chairman: J. Leon Williams, President of the Association

1. THE DENTO-ENAMEL CIRCULATION[5]

Charles F. Bödecker, D.D.S., F.A.C.D., New York City

"There is an afferent circulation in every normal tooth, passing from the pulp through the dentin into the enamel, and a return current by the same path back to the pulp chamber. . . . . The enamel-rod sheaths are in connection with the tubuli of the dentin, thus forming the end organs of the dento-enamel circulation." Microscopic demonstrations followed the presentation of data in support of this conclusion, and photomicrographs were shown. [See abstract 10, on page 217.]

[4] Meetings of sections are not included in this summary. The proceedings of the sections in 1921–27 will be published in early issues of this *Journal*. Proceedings of a sectional meeting in 1927–28 (Chicago) were published on pages 7–9 of this volume.

[5] Published in the *Journal of Dental Research*, 1926, vi, p. 113.

2. PERMEABILITY OF ENAMEL[6]

J. Leon Williams, D.D.S., L.D.S., R.C.S.I., F.A.C.D., New York City

Enamel is permeable to fluids. It is highly probable, although I do not regard this as demonstrated, that the organic fibers discovered by Dr. Bödecker constitute the medium of circulation. Our present knowledge does not warrant any definite assertion as to what the forces are which produce this circulation, nor may we assume that there is anything of order or regularity in such circulation.

The attempt has been made to show that both improvement in the quality of the fully formed enamel of erupted teeth and decay of enamel, or, at least, changes in fully formed enamel which may predispose to caries, are brought about by a fluid circulation in enamel. Not a single known fact supports such views. In my opinion further elucidation of the problem or problems of caries of enamel calls for concentration of attention and study in other directions. [See abstract 11, on page 217.]

II. SECOND MEETING: MARCH 7, 1924, CONGRESS HOTEL, CHICAGO, ILLINOIS

Chairman: Frederick B. Noyes, President of the Chicago Section

3. EPITHELIAL CHANGES IN SO-CALLED PYORRHEA

E. H. Hatton, B.L., M.D., and William G. Skillen, D.D.S., Northwestern University Dental School, Chicago, Illinois

In the histological study of material taken from persons suffering from pyorrhea our attention in the past has been concentrated on the peridental membrane, cementum, and alveolar process. One of us, Gottlieb, and others, have noticed very noteworthy changes in the epithelium. At this time we are presenting a number of specimens from which we draw the following conclusions:

(a) The pocket is quite generally lined by deformed epithelium that is fairly continuous; and, as a rule, the connective tissues are not actually denuded.

[6] Williams: *Journal of Dental Research*, 1923, v, pp. 190–93.

(b) The epithelium extends along the root for a varying distance toward the apex, and is distinctly attached to the cementum beyond the bottom of the clinical pocket.

(c) As a rule the recession of the alveolar process bears a distinct relationship to the progress of the epithelium; as the latter advances, the bone recedes in proportion.

(d) The degenerating epithelial cells attached to the cementum and in the pocket seem to furnish a matrix into which lime salts are precipitated and therefore a factor in the origin of calculus.

4. A CONTRIBUTION TO DENTAL HISTORY FROM OLD PRINTS AND ETCHINGS

William Bebb, D.D.S., Northwestern University Dental School, Chicago, Illinois

A presentation and discussion of many old and rare prints and etchings from the collection in the library and museum of the Dental School of Northwestern University.

5. STUDIES IN CONNECTIVE TISSUE TUMORS OF THE MOUTH

Frederick B. Moorehead, D.D.S., M.D., College of Dentistry, University of Illinois, Chicago, Illinois

The author discussed tumors of the mouth that consist of varying proportions of fibrous connective tissue cells, spindle cells, and giant cells. The microscopic picture is not always the best evidence of the relative malignancy of these tumors. Frequently clinical signs, such as the tendency to infiltrate and replace the bone, and to recur after operation, are more significant.

6. STUDIES OF FUSIFORM BACILLI AND SPIROCHAETES. VIII. THEIR OCCURRENCE IN ALVEOLAR ABSCESSES[7]

D. J. Davis, M.D., Ph.D., and F. B. Moorehead, D.D.S., M.D., Department of Bacteriology, College of Dentistry, University of Illinois, Chicago

Observations have been going on in our laboratory for some time upon the distribution of fuso-spirochaete organisms in the body

[7] Published in the *Journal of Dental Research*, 1923, v, p. 1. [Delayed issue.—*Ed.*]

and on lesions in which they are found. As a result of these studies it seemed that inasmuch as one of the most important localities in which these organisms are often found normally is on and about the teeth, and since they are associated with many buccal lesions, it would be reasonable to expect them to occur often and possibly play an important rôle in alveolar lesions, especially the purulent and necrotic ones. We have already shown the significance of these bacteria in a variety of conditions including pulmonary gangrene, buccal lesions, genital lesions, etc., and have called attention to their importance in determining the character of the lesions and to their association with pyogenic bacteria, particularly streptococci. Here we wish to emphasize this association of bacteria as a factor in determining the character and progress of lesions of this type in connection with alveolar abscesses.

On practically all teeth fusiform bacilli and spirochaetes and cocci, along with leptothrix threads, occur in large numbers in the bacterial masses or granules growing constantly about the gingival margin. One of the first problems, therefore, in the bacteriological study of the exudate at the terminus of a tooth would be the matter of obtaining it uncontaminated by the surrounding organisms. Upon this point great emphasis is placed.

The teeth involved in this study were removed under novocain anaesthesia by the following method: The tooth and surrounding tissue were swabbed freely with 50 per cent alcohol followed by searing the gum tissue with a hot spatula. After the tooth was removed the mouth of the wound was again seared and a platinum loop introduced into the socket. Where the granuloma remained attached to the root, it was opened with a hot spatula and a smear taken from the interior. When the granuloma remained in the socket, it was carefully removed by a small currette. Preliminary smears were made from the surface of the gum tissue and tooth previous to the extraction. These, compared with the smears taken from the socket, offer abundant proof of the differential bacteriology of the two fields.

Twenty cases of alveolar abscess have been investigated. They were practically all chronic in course and were diagnosed by x-ray examinations. In all cases indications were clear for the removal of the teeth either because of local or general lesions.

In the twenty cases in smear preparations, gram-positive cocci chiefly as diplococci or short chains were seen. They all appeared identical and had the morphology of a streptococcus, which in many of the cases were proved by culture. They were usually in moderate numbers. In all cases without exception were also seen curved gram-negative pointed bacilli of the fusiform type—usually quite numerous.

A histological study of the tissues was not attempted, but notes were made of the cellular content of the smears. In all the preparations pus cells were seen; usually they were quite numerous, though few in some specimens. Polynuclear cells were predominant as a rule. In some smears large mononuclears were fairly numerous. In two instances small granulomas attached to the end of the teeth were examined by Levaditi stain. No spirochaetes were seen in these preparations, though they contained cocci and bacilli. They were composed of chronic granulation tissue.

The material from the apical region is foul smelling and putrid, though this is at times difficult to determine with certainty, since other parts of the tooth may be putrid also. From the appearance of the material and the smear preparations, it is evident that there is also softening of a gangrenous or putrid character. Streptococcus viridans alone is ordinarily not a pus producer and does not cause putrid abscesses. The contents of these lesions, therefore, would clearly indicate that they have the characteristics of a putrid gangrenous process such as is found generally as a result of a coccal fuso-spirochaete infection.

Twenty specimens from chronic alveolar abscesses, obtained under controlled conditions, all revealed streptococci and fusiform bacilli. Sixteen revealed spirochaetes. Pus cells were commonly found.

From what is known of the relation of streptococci, fusiform bacilli, and spirochaetes, we would conclude that usually apical infections result from the combined activities of these three organisms. No doubt a variety of predisposing factors underlies the infectious process in alveolar abscesses.

III. Third Meeting: March 20, 1925: Congress Hotel, Chicago Illinois: Afternoon Session (7–8) and Evening Session (9–15)[8]

Chairmen: afternoon session—Marcus L. Ward, President of the American Association of Dental Schools and member of the Ann Arbor Section; evening session: Albert E. Webster, President of the International Association for Dental Research

7. The relation of research to teaching[8]

Edwin R. LeCount, M.D., Rush Medical College, University of Chicago, Chicago, Illinois

The author minimized the value of didactic instruction and emphasized the importance of methods that strengthen powers of independent thinking and develop the scientific attitude. "A certain amount of material, clinical material or laboratory material, or anything else to work with and putting the student to work making his own observations, piling up his own data for conclusions and for hypotheses, I think is generally regarded now as the highest type of method used in education."

8. The place of research in the university dental school[8]

Newton G. Thomas, D.D.S., M.A., College of Dentistry, University of Illinois, Chicago, Illinois

"As a teaching value, research within a school is the greatest, the best, known stimulant. . . . . Scholarship is a prerequisite to research. . . . . Dentistry has had no great scholars. It has given the world no great names: aside from their contribution to dentistry, dentists have given nothing else." Of the dentists who are teachers of dental students, but one in the United States has received the Ph.D. degree, and only one in Canada. "Six schools have not a single baccalaureate on the whole staff of specifically dental teachers.

[8] The papers by Drs. LeCount and Thomas, on the afternoon of March 20, 1925, constituted, by informal understanding, a joint program of the American Association of Dental Schools and the International Association for Dental Research. The papers, published in full in the *Proceedings of the American Association of Dental Schools*, 1925, ii, pp. 84–97, were discussed by Drs. David J. Davis, University of Illinois, and William J. Gies, Columbia University: *Ibid.*, pp. 97–101. Drs. Thomas, Davis, and Gies were and are members of the International Association for Dental Research.

. . . . What chance has a plant like research to grow in such an atmosphere. . . . . There is generally no inclination toward research on the part of men who have had only dental training, but there are a few exceptions. . . . . When the laboratory becomes a seething center of interest in the hidden things that still baffle and detain, an island of revelation in the midst of class routine and the clatter of administration, a place where the impulsion that advances the health or scholarship or wealth of the world, student imitation may be expected, but not until then. . . . . For it [dental research] we must develop scholarship that is able to see and competent to solve the problems presented in the various fields. Then we may expect research appointments and endowments. Atmosphere, the atmosphere made by doing, must be developed, and the opportunity made for student endeavor by curricular change and reward in credits for successful production. . . . . The problem is solved on a basis of intelligent appreciation of scholarship, the best obtainable, and the opportunity for its application and expression without interference or discouragement."

9. THE CRYSTALLINE PROTEINS ISOLATED FROM URINE

William H. Welker, A.C., Ph.D., Colleges of Medicine and Dentistry, University of Illinois, Chicago, Illinois

The literature of crystalline proteins of the urine was reviewed. Of these all but one are supposed to belong to the Bence-Jones group. The first crystalline protein isolated from the urine by Bramwell and Paton does not belong to the Bence-Jones group, as shown by the immunological tests made recently. Of the two crystalline proteins isolated from the urine by Dr. Hektoen, Dr. Kretschmer, and the author, one responds in general to the Bence-Jones characteristics and appears to be immunologically the same. The other, not a Bence-Jones protein, is not a blood protein, and is different from the protein of Bramwell and Paton. The identification, by means of the immunological tests, of the proteins eliminated in the urine promises to be of considerable value in diagnosis, when our knowledge as to the sources of these non-blood proteins becomes more complete. The author presented numerous lantern-slide illustrations of the forms of crystalline proteins.

10. CONCLUSIVE EVIDENCE THAT DENTAL ENAMEL CONTAINS ALBUMINOUS MATTER[9]

Charles F. Bödecker, D.D.S., F.A.C.D., and William J. Gies, Ph.D., F.A.C.D., Dental and Medical Schools, Columbia University, New York City

Bödecker's conclusion, drawn chiefly from histological data, that enamel contains protein, was confirmed by direct chemical tests of the "organic matrix" obtained by Bödecker's celloidin method of decalcification. The nature of the protein has not yet been ascertained, but the study is in progress. "Pending such a determination any discussion of the organic matrix as a factor in the nutrition of the enamel must remain speculative. . . . . If the enamel matrix is entirely inert chemically and devoid of nutritive influence, it is possible nevertheless that physical (mechanical) transmission of ions and molecules on or in its framework, distinct from physiological (selective) movement but inaugurated or controlled by conditions external to the matrix, might account for important effects on the enamel."

11. DOES THE ENAMEL OF A FULLY ERUPTED TOOTH USE NUTRIMENT OR REJECT WASTE MATTER?

J. Leon Williams, D.D.S., L.D.S., R.C.S.I., F.A.C.D., and William J. Gies, Ph.D., F.A.C.D., New York City

Nutrition is a process of selective acquisition in the structure that is being nourished. It involves a tripartite procedure—assumption, transformation, and rejection of materials—resulting in renewal of mass and in removal of waste. It is not a one-way traffic, but a process of come and go. On histological as well as chemical grounds, both phases of the question stated in the title of the paper were answered in the negative. There are no cellular nuclei in the organic matrix of the enamel to preside over a nutritive process—none to conduct selective acquisition or rejection of materials. Movement of water in enamel may occur wholly by capillarity or osmosis, without being selective in character. And, in enamel, movement of substances in

[9] Published in the *Journal of Dental Research*, 1926, vi—Bödecker, pp. 117, 131, 139; Gies, p. 143.

solution, by capillarity or osmosis, may be beneficial, indifferent, or destructive, without being nutritional.

12. ROUND-CELLED SARCOMA OF THE ALVEOLAR PROCESS[10]

Kaethe W. Dewey, M.D., and J.E. Schaefer, M.D., D.D.S., College of Dentistry, University of Illinois, Chicago, Illinois

A tumor very rarely observed in the oral cavity, a true, small round-celled sarcoma, the most malignant of all types of sarcoma, was found in a boy of twelve years in a location which is the classical seat for a benign tumor of relatively frequent occurrence; namely, the giant-cell tumor, also commonly known as epulis. In the case of the malignant tumor there was, besides the typical location, a clinical history and a gross appearance which is fairly characteristic for a certain group of these benign giant-cell tumors: a soft, spongy, vascular growth on the alveolar process occurring in children and adolescents, and developing and growing rapidly after trauma, such as a blow or the extraction of a tooth. In this boy a mass about the size of a small walnut had developed in the socket of the alveolar process two days after the extraction of a first molar; five days later it had progressed until it involved the second molar. A very malignant tumor of exceedingly rare occurrence might therefore be mistaken for a benign tumor of common occurrence.

The tumor in this case was metastatic; shortly after its removal an identical growth was found in the prostate. The patient died twenty days after excision of the latter. Histologically the tumor consisted of very closely packed, small round-cells, with nuclei slightly larger and staining more lightly than those of the small lymphocytes. The intercellular tissue was largely composed of very delicate small blood-vessels that formed the real framework for the tumor cells. The osteogenic nature of the tissue in which the tumor developed was revealed by the presence of spicules of newly formed bone in which also absorption processes could be observed.

[10] Also reported before the Chicago Pediatrics Society, April 31, 1925, and published in abstract form in the *American Journal of Diseases of Children*, 1925, xxx, p. 145.

13. PULP ABSCESSES AT THE END OF GUTTAPERCHA FILLINGS, COMPARED WITH X-RAY FINDINGS

Edward H. Hatton, B.L., M.D., Northwestern University Dental School, Chicago, Illinois

As a result of the routine examination of a considerable number of extracted human teeth with treated and filled root canals, and in which the filling did not reach to a point within two or more millimeters of the apex, a few instances have been found in which abscesses were present in the pulp stump at its junction with the guttapercha cone. In the x-ray films taken of these cases there are no regions of rarefaction at the apex. But since all these are associated with marked resorption of the dentine walls of the unfilled portion of the root canals, corresponding shadows are found by careful examination of the x-ray films. Since such an abscess may be a focus of infection, x-ray films of teeth with partially filled root canals should be studied with these facts in mind.

14. STUDY OF THE EFFICIENCY OF DENTAL FILLINGS[11]

C. Jane Fraser, M.A., University of Toronto, Toronto, Canada

This research was undertaken to determine (a) if certain of the dental cements and fillings on the market were germicidal, and (b) if any of these cements or fillings would permit bacteria to pass through or around them. *Conclusions:*

(a) The black and red copper cements were most efficient, the white ones less so.

(b) Copper amalgam is sufficiently impermeable or germicidal to be a most efficient filling, especially if it is protected on the oral surface in some way. No doubt, if left exposed for a long time, it would become permeable, as the copper would disintegrate.

(c) The silicate cements were impermeable to bacteria, but one was not impermeable to water.

(d) Guttapercha stopping is most inefficient to seal a cavity, but guttapercha, if properly put in, should be quite satisfactory.

[11] Published in the *Dominion Dental Journal*, 1925, xxxvii, p. 1.

(e) The silver cements require very careful technique in mixing and insertion.

15. COPPER AMALGAM AS A ROOT-FILLING MATERIAL

F. C. Husband, D.D.S., Toronto, Canada

Copper amalgam has been known to the dental profession for many decades. A number of authorities have recorded their observations on its qualities during this time—noting particularly its antiseptic properties, not only when freshly prepared, but in old fillings. Miller observed that the dentin of extracted teeth which had been filled with copper amalgam was not only sterile, but had antiseptic properties. Fraser further found that, of all filling materials available to dentistry, copper amalgam *alone* possessed *permanent* antiseptic properties. This suggested its use in the filling of root canals. Its insertion involves thorough surgical and medicinal preparation of the canal, and guiding it to place with flat-ended pluggers. From 3 to 5 mm. of the apical portion is thus filled, and the remainder with oxychlorid of zinc into which a guttapercha cone is pressed. Anatomical features of the root end are such—multiforamina, etc.—that one of three things must be accomplished to make a root-canal operation a success:

(a) Induce, if possible, physiological closure of these foramina (Davis).

(b) Mechanically obliterate (or fill) them.

(c) Maintain asepsis of a permanent character in that area without causing irritation of the periapical tissues.

Briefly consider them in order:

(a) *Physiological closure.* Copper amalgam placed close to the apical end of the canal, following careful preparation, will permit and not hinder this repair, because of its permanent germicidal and non-irritating properties.

(b) *Mechanical obliteration.* This is well-nigh physically impossible, except in roots with a single foramen.

(c) *Maintenance of asepsis.* Copper amalgam is the only filling material we have which possesses permanent antiseptic properties. Further, it penetrates the tooth tissue, rendering it antiseptic.

Copper amalgam seems to offer qualities which meet the need as no other material does, while clinical experience has amply justified our highest expectations.

IV. Fourth Meeting: March 26, 1926, Congress Hotel, Chicago, Illinois

Chairman: Frederick B. Noyes, President of the Association

16. Preliminary Report on Clinical Research Data for the Control of Caries

Harold DeWitt Cross, D.M.D., F.A.C.D., Forsyth Dental Infirmary, Boston, Massachusetts

The clinical procedures resulting from a plan of early-age attention have proved to be so efficient that upon them has been placed the chief reliance for the control of caries, rather than the cleaning of the teeth. In like manner, "cleaning" has been replaced by attention to "nutrition," determined by the condition of the teeth, the weight and general condition of the child as noted from a physical examination, and from a food, habit, and endocrine check-up.

In 1915, one in every three teeth extracted was a first molar, or 33.3 percent. This has been reduced to one in every two hundred, or 0.5 percent, in groups of children who have received this early-age treatment.

Attempts to correlate weight and height to tooth conditions, on the grounds of the nutrition factor, have not been very encouraging. In a group of six hundred children, it was found that the percentage of good or poor teeth was in no way influenced by either over-weight or under-weight, as compared with the percentage in normal weights. Even when using as high as 20 percent over-weight and 10 percent under-weight, results were about the same. This, however, may not be an indication of the lack of connection between tooth conditions and nutrition, but an indication rather that nutrition is not based entirely upon weight, and that many other factors must be considered.

Similar attempts to correlate data as to the type of food in regard to the acid or base residue have been wanting in specific indications of a definite relation to active caries, although thus far the data pertain to

cases which have not been carried out with sufficient detail and number to warrant any definite conclusions.

Data collected from a definite group of 13,000 cases as to the number of first permanent molars extracted, in relation to the initial-treatment age of the children, shows that the number of extractions increases in definite ratio to the age. Two were extracted for five-year, eight for six-year, eleven for seven-year, and sixteen for ten-year cases. During this time, similar extractions for promiscuous cases of approximately the same ages, but not treated early, included 730 first permanent molars.

17. STUDIES OF THE RELATION OF BACILLUS ACIDOPHILUS TO DENTAL CARIES[12]

Russell W. Bunting, D.D.Sc., F.A.C.D., School of Dentistry, University of Michigan, Ann Arbor, Michigan

In a survey of the oral microbic flora as it relates to the occurrence of dental caries the author, in collaboration with an associated group, has examined approximately 3000 cases, and found that in them there was a very close correlation between the presence or absence of B. acidophilus and the activity of dental caries. So definite is this correlation that, in the opinion of this group, the presence or absence of B. acidophilus in the mouth constitutes a definite criterion of the activity of dental caries that is more accurate than any clinical estimation can be. Furthermore, it was noted that there was a spontaneous cessation of caries coincident with the disappearance of B. acidophilus from the mouth, either from prophylactic, therapeutic, or dietetic control. Caries-free individuals who upon first examination had B. acidophilus overgrowths in the mouth, later developed active caries. It was found that the degree of dental caries, and the activity of B. acidophilus in the mouth, are apparently related to the type of diet received and the form of dental attention afforded.

[12] Subsequent publications covering and extending this work: (a) The rôle of Bacillus acidophilus in dental caries; Bunting and Palmerlee, *Journal of the American Dental Association*, April, 1925, p. 381. (b) Further studies of the relation of Bacillus acidophilus to dental caries; Bunting, Nickerson, and Hard, *Dental Cosmos*, October, 1926, p. 931. (c) Further studies of the relation of Bacillus acidophilus to dental caries (II); Bunting, Nickerson, Hard, and Crowley, *Dental Cosmos*, January, 1928, p. 1.

18. THE ACTION OF GERMICIDES IN THE PULP CANAL, OR THE POWER OF DISINFECTANTS TO STERILIZE DENTINE

C. Jane Fraser, M.A., and A. E. Webster, M.D., D.D.S., F.A.C.D., University of Toronto, Toronto, Canada

The purpose of the research was to determine the action of certain germicides in the pulp canal, and to ascertain whether bacteria can pass through the apical foramina. *Conclusions:*

(a) Bacteria pass readily from the canal to the apical area. The infection of the apical area from an infected canal is almost unavoidable.

(b) The efficiency of the germicides that were tried in the root canal was about 50 per cent.

(c) The ability of germicides to pass through the apical foramina, and thus to sterilize the apex, is most uncertain.

19. THE RELATION OF NASMYTH'S MEMBRANE TO PERIODONTOCLASIA[18]

Kurt H. Thoma, D.M.D., Department of Oral Histology and Pathology, Harvard University Dental School, Boston, Massachusetts

The author presented photomicrographs showing conditions at the base of the gingival crevice, and the manner in which the crevicular epithelium is attached to the tooth. At the base of the gingival crevice, the epithelium gives attachment to Nasmyth's membrane (enamel cuticle), which completes the protective lining of the gingival crevice. "Periodontoclasia . . . . may be prevented to a great extent by preserving the crevicular tissue. It seems not at all unlikely that the use of abrasives on the rubber wheel, brush wheel, or orangewood stick by the dentist, or on the toothbrush by the patient, may tear away or injure the enamel cuticle under the gum margin, breaking the continuity of the protective membrane lining the gingival crevice. While disease may not follow at once, such injury certainly represents a permanent defect, which leaves a path open to infection." The wisdom of scaling tooth surfaces with sharp instruments in normal mouths should also receive thought in the light of the presented facts.

[18] Published in the *Journal of Dental Research*, 1926, vi, p. 7.

20. NUTRITION STUDIES OF DENTAL IMPORT

Sam Karlström, D.M.D., and William J. Gies, Ph.D., F.A.C.D.,
Laboratory of Biological Chemistry, Columbia University,
New York City

(*A*) *Does the excessive ingestion of milk favor deposition of salivary calculus?*[14]

Milk contains, or in its assimilation yields, a large proportion of earthy phosphate, the chief ingredient of salivary calculus. An effort was made to determine whether the ingestion of milk in abundance, as often occurs under modern dietary recommendations, might be attended by increased formation of hard tartar. The experiments, which thus far have been preliminary only, were restricted to analyses of saliva obtained from a man 29 years old. He was in good health, and had normal gums and no pyorrhea. Tendency to tartar formation was very slight, and for several years there had been few new signs of dental caries. Three daily dietetic conditions were compared during a period of 51 days: (a) normal diet; (b) normal diet, plus an extra liter of water; (c) normal diet, plus an extra liter of milk. The effects of these conditions were compared on a daily routine that was uniform in all other respects. Suitable intervals and alternations prevented overlapping of effects. Specimens of saliva were obtained at the same relative periods and by mechanical stimulation (chewing pure paraffin).

Familiar variations in composition, similar to those of saliva on any physiological basis, were noted, but there was no significant increase in the content of calcium, phosphorus, or calcium phosphate. There were no indications of any increase in tartar formation. The senior author's plan includes extensions to apply to conditions at middle age, including persons in good health and others having different degrees of periodontoclasia.

[14] Published in the *Svensk Tandläkaretidskrift*, 1927, xx, p. 190. (Included in a dissertation by Dr. Karlström, now resident in Gävle, Sweden.) The experiments were conducted in 1923.

(*B*) *Do the conditions of pregnancy and lactation affect the chemical composition of the teeth in animals continuously on a normal diet?*[15]

Twenty-seven female white rats, in five groups, were given an adequate balanced diet. The breeding conditions with three males (brothers) and female controls (sisters—two or three in each group) were made strictly comparative for the individuals in each group. The resultant litters were normal in size and condition. The analyses were restricted to the incisor teeth. Three sets of mothers and controls were killed fifteen or thirty days after the birth of the litters—two groups, immediately afterward.

There was no discernible effect on the percentages of organic matter, inorganic matter, calcium, phosphorus, or calcium phosphate, in the teeth of the mothers as compared with those of the control females.

Negative results for the incisor teeth of white rats, which grow rapidly throughout the whole life of the individual, may not be applicable to human teeth, although it was believed that the conditions of continuous growth, with consequent greater relative dental metabolism, might yield striking evidence of relationship.

(*C*) *Does a diet deficient in vitamins, before and during pregnancy, have a harmful influence upon the composition of the teeth of the offspring that cannot be rectified by a normal diet for the mother during the period of lactation?*[16]

Three series of breeding experiments were conducted on growing adult white rats—eight animals from one or two litters in each series—divided equally into control and "tested" sub-groups. After normal conditions had been maintained during a preliminary period, the control females were continued on the adequate diet, but the tested females were given a "synthetic" diet prepared from these ingredients in the proportions indicated:

Casein—heated for 48 hours at 105°C. before use, 25 gm.; *starch*,

[15] Published in the *Svensk Tandläkaretidskrift*, 1927, xx, p. 176. (Included in a dissertation by Dr. Karlström.) The experiments were conducted in 1922–23. See footnote 14.

[16] Published in the *Svensk Tandläkaretidskrift*, 1927, xx, p. 145. (Included in a dissertation by Dr. Karlström.) The experiments were conducted in 1923. See footnote 14.

53 gm.; *lard*, 24 gm.; *salt mixture*, 4 gm.; *water*, 300 cc. The *salt mixture* consisted of tri-basic calcium phosphate, 10 gm.; potassium mono-hydrogen phosphate, 37 gm.; sodium chloride, 20 gm.; sodium citrate, 15 gm.; magnesium citrate, 8 gm.; calcium lactate, 8 gm.; ferric citrate, 2 gm.

After the increasing weight of the tested animals began to lag, because of the vitamin deficiency in the diet, the breeding was begun. In each series one male rat was transferred daily from the control sub-group to the tested sub-group, or vice versa; and as the earliest indications of pregnancy were noted, the affected animals were isolated. Immediately after the birth of a litter, the diet of each mother in the tested sub-group was changed to the adequate diet of the control sub-group. Thirty days after birth the litters were killed and their incisor teeth subjected to analysis for organic matter, inorganic matter, calcium, and phosphorus.

There were no significant differences for the teeth of the offspring of tested rats as compared with those of the related control rats. It was evident, also, that mothers which shortly before and during pregnancy received a vitamin-deficient diet, but after bearing the litters were fed a normal diet, reared the young rats as effectually as did the control mothers. The average number in the litters of the tested rats was smaller, however, than for the control rats, and the number of still-born was larger, exceptions that materially qualify the significance of the findings. The litters of a number of tested mothers that had been continued on the diet deficient in vitamins (after the birth of the litters) lived for only a few days.

The comment in the last paragraph of abstract (B), above, also applies to the results of these experiments.

V. Fifth Meeting: April 19, 1927, Hotel Plaza, New York City

Chairman: Leuman M. Waugh, President of the Association and Secretary of the New York Section

21. Preliminary Report on the Elastic Elements of the Peridental Membrane[17]

Newton G. Thomas, M.A., D.D.S., Department of Anatomy, Dental School, University of Pittsburgh, Pittsburgh, Pennsylvania

Using Weigert's resorcin fuchsin stain and jaws of dogs, the author found elastic fibers, contrary to prevailing belief, scattered throughout the peridental membranes of all the teeth. They were abundant, fine, and arranged usually in the plane of the white fibres, which they sometimes accompanied, but they also showed complete absence of arrangement in place. They did not appear in the tissue in contact with either tooth or bone.

22. Bacteria in the Vital Pulps of Human Teeth[18]

Andrew J. McDonagh, L.D.S., Toronto, Canada

The pulps of about 65 per cent of thirty-four vital teeth, perfectly covered by cementum and enamel, and extracted and examined with all necessary scientific precautions at St. Michael's Hospital, Toronto, contained bacteria. "The theory that perfect teeth do not contain microörganisms, and that bacteria enter the pulp only when enamel is ground off, or through cavities, or by way of pockets, was not borne out by the investigation." The organism usually found was Streptococcus viridans, in about one-third of the cases Staphylococcus aureus, but in one case Bacillus coli communis. The latter seems to have been derived from a primary focus elsewhere, probably from an appendiceal abscess removed from the patient two weeks previously. In many instances, where two apparently perfect teeth in the same mouth were extracted under identical conditions, the pulp of one contained bacteria but the other did not. In some cases the patient gave a history of having no constitutional trouble, and yet there were microörganisms in the pulps of some of the perfect teeth. "Would

[17] Published in the *Journal of Dental Research*, 1927, vii, p. 325.

[18] Published in the *Journal of Dental Research*, 1927, vii, p. 411.

one not be justified in concluding. . . . that [in some cases] the source of selective organisms in septic areas surrounding, and in roots of, teeth may be a long distance from the teeth?" Pulps taken from several hundred teeth having only small cavities, or slight abrasions or stains, were found to contain the same kind and about the same proportion of microörganisms as the pulps of perfect teeth. The study is in progress.

23. EFFECTS OF "TAXI" AND OF "STAIN REMOVER," SO-CALLED TOOTH-CLEANING FLUIDS, ON DENTAL ENAMEL[19]

Charles F. Bödecker, D.D.S., F.A.C.D., Dental School, Columbia University, New York City

These corrosive solutions of strong mineral acids, when applied to perfect enamel surfaces for thirty seconds to nine minutes, invariably dissolved considerable material. Numerous photomicrographs presented convincing evidence that neither product can be safely used in dental practice. When cleaning fluid on a cotton pellet is applied to teeth, it does not remain on the labial and buccal surfaces, but by capillarity quickly passes to the interproximal surfaces, which, on account of their inaccessibility, cannot be well polished and are thus rendered more susceptible to decay. The most cautious application of a tooth-cleaning fluid, with intent to restrict contact to a limited surface for only a few seconds, cannot prevent undesirable extensions of the period of chemical action. This is not only true of unavoidable delays in removing the fluid, but also applies to areas which, by their inaccessibility, retard or prevent prompt removal of the acid mixture, which then may corrode the enamel to the full extent of its destructive power. "The use of these fluids in dental practice is contraindicated." [See the succeeding abstract.]

[19] Published in the *Journal of Dental Research*, 1927, vii, p. 480.

24. THE ACIDITY OF "TAXI" AND OF "STAIN REMOVER," SO-CALLED TOOTH-CLEANING FLUIDS[30]

Maxwell Karshan, M.A., Ph.D., Dental and Medical Schools, Columbia University, New York City

Each of these fluids contains a large proportion of strong mineral acid, chiefly hydrochloric. Fluoride and some hydrofluoric acid (by hydrolysis?) are present in "Stain Remover." Each fluid, within 30 seconds, dissolves "considerable material" from enamel. The data for acidity, which explain this result and also those in the last preceding abstract, are assembled in this summary:

SOLUTION	pH	H-ION CONCENTRATION	CC. OF 0.1 N NaOH TO NEUTRALIZE 5 CC. OF FLUID	CALCULATED NORMALITY OF ACIDITY	PERCENTAGE STRENGTH EXPRESSED IN TERMS OF HCl
"Taxi"	0.070	0.85 N	57.1	1.1	4.0
"Stain Remover"	0.017	0.97 N	63.0	1.3	4.8

[SIXTH MEETING: MARCH 25, 26, 27, AND 28; WASHINGTON, D. C. Proceedings to be published in an early issue of this *Journal*.]

C. MEMORANDUM ON EXECUTIVE PROCEEDINGS

A summary of the executive proceedings for 1920–28 will be published in the Supplement of this volume, to be included in the number for December, 1928.

D. LIST OF OFFICERS AND MEMBERS (MARCH 15, 1928)

A. OFFICERS: ELECTED AT THE ANNUAL MEETING IN 1927

President: Leroy M. S. Miner, Harvard University
Vice-president: Russell W. Bunting, University of Michigan
Secretary: Albert E. Webster, University of Toronto
Treasurer: William Rice, Tufts College
Honorary President: William J. Gies, Columbia University
Council (three representatives of each of the six sections):
Ann Arbor Section—Russell W. Bunting, U. G. Rickert, Marcus L. Ward

[30] Published in the *Journal of Dental Research*, 1927, vii, p. 496.

Boston Section—V. H. Kazanjian, Kurt H. Thoma, George H. Wright
Chicago Section—William Bebb, Edward H. Hatton, W. H. Welker
New York Section—Arthur H. Merritt, Bissell B. Palmer, Jr., Leuman M. Waugh
San Francisco Section—John A. Marshall, Guy S. Millberry, John S. Shell
Toronto Section—A. J. McDonagh, Harry S. Thomson, Albert E. Webster

Committee on Organization—William J. Gies, Leroy M. S. Miner, and Leuman M. Waugh

B. Members

(The list does not include the new members elected at the Sixth Annual Meeting in Washington, D. C., on March 25–28, 1928)

1. Ann Arbor Section

R. W. Bunting, Dental School, University of Michigan, Ann Arbor, Michigan
C. J. Lyons, Dental School, University of Michigan, Ann Arbor, Michigan
*U. G. Rickert, Dental School, University of Michigan, Ann Arbor, Michigan
E. O. Scott, Dental School, University of Michigan, Ann Arbor, Michigan
Marcus L. Ward, Dental School, University of Michigan, Ann Arbor, Michigan

2. Boston Section

Lawrence W. Baker, 230 Beacon St., Boston, Massachusetts
*Fred R. Blumenthal, 475 Commonwealth Av., Boston, Massachusetts
Walter G. Bridge, Tufts Dental School, Boston, Massachusetts
F. P. Chillingworth, Tufts Dental School, Boston, Massachusetts
Harold DeWitt Cross, Forsyth Dental Infirmary, Boston, Massachusetts
Adelbert Fernald, 420 Boylston St., Boston, Massachusetts
Percy R. Howe, Forsyth Dental Infirmary, Boston, Massachusetts
A. Leroy Johnson, 576 Fifth Avenue, New York City[21]
V. H. Kazanjian, 475 Commonwealth Av., Boston, Massachusetts
L. M. S. Miner, 363 Marlboro St., Boston, Massachusetts
Arthur L. Morse, 283 Dartmouth St., Boston, Massachusetts

* Secretary of the Section.
[21] One of the founders of the Boston Section, who continues his membership in that Section.

William Rice, Tufts Dental School, Boston, Massachusetts
Alfred P. Rogers, 520 Beacon St., Boston, Massachusetts
Kurt H. Thoma, 43 Bay State Road, Boston, Massachusetts
George H. Wright, 149 Newbury St., Boston, Massachusetts

3. CHICAGO SECTION

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Thomas L. Gilmer, 180 N. Michigan Av., Chicago, Illinois
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F. B. Moorehead, 30 N. Michigan Av., Chicago, Illinois
Frederick B. Noyes, 30 N. Michigan Av., Chicago, Illinois
Victor T. Nylander, 1836 W. Harrison St., Chicago, Illinois
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Newton G. Thomas, Pittsfield Bldg., Chicago, Illinois
W. H. Welker, University of Illinois, 508 S. Honore St., Chicago, Illinois

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Theodor Blum, 101 East 79th St., New York City
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H. S. Dunning, 33 East 68th St., New York City
W. B. Dunning, 140 East 80th St., New York City
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William K. Gregory, American Museum of Natural History, New York City

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Arthur H. Merritt, 58 West 47th St., New York City
Henry Fairfield Osborn, American Museum of Natural History, New York City
*Bissell B. Palmer, Jr., 667 Madison Av., New York City
M. L. Rhein, 38 East 61st St., New York City
J. P. Ruyl, 285 Madison Av., New York City
M. I. Schamberg, 952 Fifth Av., New York City
Paul R. Stillman, 52 Vanderbilt Av., New York City
L. M. Waugh, 576 Fifth Av., New York City
J. Leon Williams, 160 Riverside Drive, New York City
J. Lowe Young, 18 West 74th St., New York City

5. SAN FRANCISCO SECTION

George L. Bean, Fitzhugh Building, San Francisco, California
John A. Marshall, Dental School, University of California, San Francisco, California
Edwin H. Mauk, Dental School, University of California, San Francisco, California
Guy S. Millberry, Dental School, University of California, San Francisco, California
Forrest H. Orton, Dental School, University of California, San Francisco, California
*John S. Shell, Dental School, University of California, San Francisco, California
F. V. Simonton, Dental School, University of California, San Francisco, California

6. TORONTO SECTION

J. Frank Adams, 54 College St., Toronto, Canada
G. R. Anderson, 7 Rose Park Crescent, Toronto, Canada
Harold K. Box, Dental School, University of Toronto, Toronto, Canada
A. J. Broughton, 86 Bloor St., West, Toronto, Canada

* Secretary of the Section.

*W. E. Cummer, Dental School, University of Toronto, Toronto, Canada
A. W. Ellis, 67 Yonge St., Toronto, Canada
C. Jane Fraser, Toronto University, Toronto, Canada
Andrew Hunter, Toronto University, Toronto, Canada
F. C. Husband, 2 Bloor St., East, Toronto, Canada
Andrew J. McDonagh, 2 Bloor St., East, Toronto, Canada
E. W. Paul, 205 Yonge St., Toronto, Canada
Harry S. Thomson, 18 Summerhill Gardens, Toronto, Canada
W. C. Trotter, 326 Bloor St., West, Toronto, Canada
A. E. Webster, Dental School, University of Toronto, Toronto, Canada

7. NOT NOW FORMALLY MEMBERS OF ANY SECTION

Kaethe W. Dewey, Dental School, University of Pittsburgh, Pittsburgh, Pa. (Formerly Chicago)
Roscoe H. Volland, First National Bank Building, Iowa City, Iowa

Total number of active members (March 15, 1928)..... 81

8. RESIGNED (1920–28)

Guy M. Clark, San Francisco; *Cyrenus G. Darling*, Ann Arbor; *Ludvig Hektoen*, Chicago; *H. G. Wells*, Chicago.

9. DECEASED (1920–28)

R. R. Andrews, Boston; *George A. Bates*, Boston; *E. A. Bogue*, New York; *J. J. Mackenzie*, Toronto; *Frederick A. Peeso*, New York; *E. H. Smith*, Boston; *H. Carlton Smith*, Boston; *Eugene S. Talbot*, Chicago; *F. T. Van Woert*, New York.

* Secretary of the Section.

THE MOST SIGNIFICANT FINDINGS OF THE CALIFORNIA STOMATOLOGICAL RESEARCH GROUP IN THE STUDY OF PYORRHEA[1]

F. V. SIMONTON, D.D.S.

San Francisco, California

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I. Introduction

It is a privilege to appear before you this evening to talk upon a subject of great mutual concern. Communications I have received recently from and through Professor Gies intimate that your interest in research would not be satisfied by a mere enumeration of data. I am led to believe that you feel as we do that support given dental research is a trust of concern to the entire profession, and hence your

[1] Presented at a joint meeting of the New York Section of the International Association for Dental Research and the New York Academy of Dentistry, at the Commodore Hotel in NewYork City, on December 14, 1927.

attitude involves participation. This is very gratifying. We have always welcomed, and as far as possible sought, suggestions and criticisms.

I have decided, even at the risk of taxing your time and patience, to present a report of our work in such manner as seems best fitted to acquaint you with the deeper nature of our responsibilities; our points of view and orientation; our accomplishments both in the light of these factors and in their bearing upon practical therapeutic procedure. If I succeed in making clear to you what we are endeavoring to accomplish; elucidate as briefly as possible our results and point out their significance as we see it; explain the machinery and methods of operation of our organization, and lay before you an evaluation of our assets and liabilities as seen by one intimately, actively, and responsibly concerned, we will then have established a basis upon which we can coherently and intelligently discuss the situation, and upon which we can most effectively utilize the past to plan the future. I feel that such a discussion, with the type of men who are present, would in itself constitute a real contribution and prove of value to future research, wheresoever and by whomsoever it may be carried on.

II. Illustrations of the Backwardness of Biologic Research in Dentistry

A student program was presented at a dental fraternity house in San Francisco in 1912. A senior student read a paper compiled from the literature. The data selected portrayed pyorrhea as a disease of the investing tissues of the teeth associated with deposits of tartar. A physician present, instructor in bacteriology and pathology, stated in effect that it was apparent that nothing of significance was known of the cause of the disease, that the investigation of the etiology was a medical problem, that the physicians at that time could contribute nothing, and that the dentist had therefore gone as far as he could.

There are two species of dentistry. The European species is an organism; the American, a mechanism. There are fossil remains of an organic species that existed in America about twenty years ago; possibly it migrated to this continent; but it may be only pseudo-dental and really a member of a different genus—the medical.

In 1912, Noyes' "Dental Histology" was introduced as a text book

in our school. No associated laboratory work was possible, as the only dental specimens available were a few sections of developing teeth belonging to the department of general histology. In 1914, the instructor in dental histology introduced a laboratory course with specimens prepared with the assistance of a technician, who taught him histologic technic in exchange for instruction in dental mechanics. A few sections of the investing tissues were included in this collection, and the "instructor" for the first time saw their structure under a microscope. In 1919 a biopsy was accomplished at San Quentin Prison, and a specimen in an advanced state of pyorrhea was obtained. A year later, one of the prominent pyorrhea specialists in our city, having heard that we had such material, asked to see a specimen under the microscope. After looking at it he remarked that it did not mean much to him because it was so long since he had looked at such a specimen. The truth was that he did not realize that *he had never seen under a microscope a specimen, either normal or diseased, of the tissue in the treatment of which he was a specialist.* As recently as a few months ago one of our specialists, visiting in the East, called on a prominent dental pathologist and not only confessed that he had never seen dental tissues under a microscope, but stated further that he was not interested in seeing them.

In response to a criticism that we were neglecting the teaching of prophylaxis, we conferred at length with two pyorrhea specialists, the leading protagonists of two schools of thought—the one advocating gentle conservative instrumentation, the other vigorous instrumentation. We favored the former, and in our subsequent teaching varied and elaborated its *technical detail*.

The writer was recently asked to review a new edition of a well known text on dental pathology, and was astonished to find that the great wealth of literature on histopathology in the German language had been ignored. What argument could better prove the need for scientific biologic research in dentistry than a consideration of such local but doubtless characteristic circumstances and events, together with a review of the "available" literature. (European literature was not, and to a large extent is not, "available" to a profession which does not regard acquaintance with a foreign language as practical.)

And so an urge developed to initiate, to awaken, interest in or to

inspire the desire for, the planning or the building of an institute of biology amidst the factories and physico-chemical laboratories of dentistry. The urge was not indigenous to this locality alone, for the fruits of the last several decades indicate that similar antecedent and contemporaneous *Ursprünge* occurred in other parts—in New York as early as 1909.

III. Reattachment

When we undertook to evaluate the views of different clinicians, we recognized that the problem of "reattachment" was of critical importance. It occurred to us that the rational thing to do was to excise a a block of treated tissue and see what the conditions following treatment actually were. We first sought for a case where the biopsy removal of pyorrheal tissue would be justified in a prescription for partial denture restoration. We excised from such a case, for control purposes, two upper bicuspids and their investing tissues and prepared this material for microscopic study. We then sought out a similar case; and, after treating by scaling and hygiene a corresponding region for nine months, excised tissue as before and prepared sections.[2] We have since repeated this experiment three times, but have delayed reporting on it in order to secure and study still further material.

In 1912 McCall showed at a meeting of the Alumni Association of the University of Buffalo a case which he had treated. A tooth with its investing tissues was subsequently excised from this case, in 1920, and prepared and studied microscopically by Box. In one photomicrograph is shown the region of the cemental gingiva. An *Epithelansatz* covering an irregular surface of the cementum appears, which can be interpreted as reattachment. We have particularly convincing reattachment of this character, in one case. In a photomicrograph of a region apical of the *Epithelansatz* a rather shallow excavation of the older cementum appears, and new cementum covers it. Box interprets this as a repair following instrumental removal of tissue in scaling. Gottlieb[3] points out that this is more probably a

[2] Hanford, W. H., Patten, C. O., Westbay, C., and Simonton, F. V. A contribution to our knowledge of pyorrhea from the standpoint of histopathology. *Dental Cosmos*, 1923, v. 65, pp. 12–17.

[3] Gottlieb, B. Paradental pyorrhea and alveolar atrophy. *Fortschritte der Zahnheilkunde*, 1926, p. 371.

resorption and repair such as occurs commonly in untreated tissue. The fact that there are no embedded Sharpey's fibres in this region, but that the fibres run longitudinally, does not prove that this region received instrumental treatment. We showed, in "Etiology of Parodontoclasia" (Fig. 66),[4] that untreated tissue may possess the same appearance. It would seem that, since there are no cementoblasts to be observed adjacent to the new cementum mentioned, the resorption or curettement must have taken place previous to the occurrence of the condition of the pericementum as shown. The absence of epithelial rests does not, as Gottlieb points out, prove that this region was curetted, since their presence and position are variable. Again, the process which led to alteration of the arrangement of the pericemental fibres may have dispersed them.

The x-ray pictures of the case of Box taken at an interval of a year before and a year after treatment are not convincing, first, because the septa adjacent to the one that appears restored are not affected (I presume the treatment was not restricted to one tooth); secondly, because the radiographs[5] are not comparable, as they are of different densities. There is a trace of septum in this region, at the same level in the untreated condition as in the treated one.

Apropos of the adequacy of proof of reattachment offered by McCall and Box, Gottlieb says:[6]

> Leider haben McCall und Box die ursprüngliche Taschentiefe vor der Behandlung nicht mit objektiver Sicherheit festgestellt. Täuschungen über die ursprüngliche Taschentiefe sind da nur sehr leicht denkbar.
>
> "Ich glaube nicht, dass wir mit diesen Autoren uns dem Glauben werden hingeben können, dass die pyorrhoe in dem Sinne eine heilbare Erkrankung sei, dass wir durch konservierende Behandlung in der Regel den Taschenboden wieder an die Schmelz-zementgrenze verlegen können."

Gottlieb also points out (pp. 376–377) that resorption and consolidation of bone, such as shown in Box's radiographs, can be induced by endogenous factors having nothing to do with treatment.

[4] Simonton, F. V. The etiology of parodontoclasia. *Journal of the American Dental Association*, 1927, v. 14, pp. 1767–1828.

[5] Box, Harold Keith. Studies in periodontal pathology. 1924. *Canadian Dental Research Foundation, Bulletin No. 7*, pp. 77 and 78.

[6] Gottlieb: Paradental pyorrhea and alveolar atrophy. *Fortschritte der Zahnheilkunde*, 1926, pp. 371–372.

It seems well to postpone formal statement on this subject until we have studied our new material for a long enough period of time to make our best attempt at interpretation; and possibly, also, until we repeat the experiment several more times. However, because of the great interest in this subject, I am willing to state my personal views. I believe that in certain cases reattachment will take place through the epithelium which covers the treated crevicular surface uniting with the cementum along an *Epithelansatz*. The type of case which appears most promising for this result is one with vertical lesions and marked vascular reaction.

New bone growth is apparent in all our material, but it does not necessarily represent additional bone. I have evidence in one of my cases that there is slightly less bone than before treatment. The improvement of conditions by the treatment and stimulation led to a removal of some of the crest of the old bone and repair by new, leaving the bone in a better physiologic condition but with its crest at a slightly receded level. Movements that may take place in the treated teeth due to improved tone lead to new deposition of cementum. As a result of study of this material and other observations which will be mentioned later, I have developed an impression that pyorrhea is not necessarily a continuous process, but that it may lie dormant or become aborted. In the case referred to in this paragraph, I believe the pyorrhea was dormant.

Apropos of this problem, I have observed, in experimentally traumatizing the pericementum of the dog, that infection and resorption first occur, followed by healing, with attachment at a level *further apical*. Before this problem of reattachment can be definitely settled, we shall have to do further work, taking into consideration more accurate diagnosis, for all pyorrhea is not the same; and we may easily be led into the mistake of treating in the same way, and expecting similar results from, types of pyorrhea of radically different or mutually exclusive causation. Fortunately, we are steadily approaching what will probably serve as an adequate method of diagnosis. There is also the somewhat simpler matter of perfecting a "water tight" method of determining precisely where the scaler has actually passed. Gottlieb recommends a fine point inserted to the depth of the pocket, and radiographed before and after treatment.

The difficulty here is in getting the point in the same position each time. We have made considerable study of methods of revealing the outline of the attachment—injecting bismuth paste was one of them—but finally designed an apparatus to be described subsequently, by which it is possible to insert a point in the same position each time. In our published statement of work on this problem we drew the following conservative conclusions: that instrumental treatment of pyorrhea is beneficial; that some regeneration takes place; that the histologic picture of the treated tissue is essentially constructive while that of the diseased tissue is essentially destructive, and that the inflammation in the former is markedly reduced.

IV. Local Microbiology as Affected by Treatment

We next became concerned about the effect of treatment upon the microbiology. This study was desirable in order to gain information upon the degree and rate at which sepsis might be lessened, that we might learn further of the effectiveness of treatment both as to healing and control of sepsis as a possible source of focal infection. The study was approached by two different methods in the hands of two different workers and conformable results obtained. A. P. Batchelder,[7] in a study of the relation of microörganisms to pyorrhea and its treatment (unpublished manuscript), obtained the following results (Table 1):

(1) A marked diminution of the bacterial flora, both in morphologic variety and in total numbers, occurs in a few weeks through scaling and hygienic procedure.

(2) The spirals, fusiform bacilli, and very small, fine bacilli experienced the greatest reduction.

(3) The cocci and *B. subtilis* were least reduced.

(4) The organisms most refractory to artificial culture were the most affected by treatment.

(5) Some reduction occurred about the control teeth due quite possibly to the inoculating effect of the instrumentation.

[7] The method used by Mr. Batchelder was that of direct smears and percentage counts, together with cultural procedures. Dr. Beckwith's similar findings were made by an entirely different method, *i.e.*, by the use of the dark-field microscope.

T. D. Beckwith[8] showed that during the course of treatment by scaling, the spiral forms diminish first, the larger rods next, and the

TABLE 1

Data concerning microörganisms associated with teeth under several methods of treatment, and control teeth. (From an unpublished manuscript by A. P. Batchelder)

ORGANISM	AVERAGE NUMBER OF ORGANISMS PER FIELD				
	Beginning of treatment	End of 2nd week	End of 5th week	End of 6th week	End of 8th week
Patient 35210. Tooth: Upper left 1st molar; treated by scaling alone					
Spiral[1]	34	29	21	22	16
Streptococci	26	26	18	16	14
Staphylococci	21	20	18	16	13
Long bacilli	15	21	22	21	9
Fusiforms	9	9	9	9	4
Short bacilli	8	11	11	11	8
Diplococci	2	0	1	1	0
Patient 35210. Tooth: Lower left 2nd molar; treated by scaling and application of acriflavine					
Spiral[1]	29	23	15	12	
Streptococci	27	24	13	14	
Staphylococci	20	17	12	11	
Long bacilli	15	12	5	8	
Short bacilli	8	8	7	6	
Fusiforms	5	8	5	5	
Diplococci	5	2	1	0	
Patient 35210. Tooth: Upper left 2nd molar. No treatment—control					
Spiral[1]	29	17	22	24	
Streptococci	25	27	16	22	
Staphylococci	21	24	16	17	
Fusiforms	16	17	20	21	
Long bacilli	15	16	11	13	
Short bacilli	11	11	10	10	
Diplococci	9	12	5	8	

[1] Note the predominance of this organism in each case.

micrococci follow. These studies, aside from the suggestion of the value of a spirochetocide, have no application as expressed by technical

[8] Beckwith, T. D. The dark field microscope in pyorrhea and oral infections, *Journal of the American Dental Association*, 1925, v. 14, pp. 1228–1230.

procedure, but are none the less practical. It is of great practical importance to know how much reduction in sepsis can be expected in a certain time. A dentist might find it very important, and be rather well pleased to be able to say in consultation with a physician, that he could accomplish a certain amount of reduction of sepsis in a given time in a case where such reduction was urgent.

The observation that the control teeth improved suggests that anti-bacterial treatment may be of benefit. Little support for this form of treatment has been afforded by our general clinical observation. However, as Appleton says, it is logical to expect that vaccines have some value. They may prove important as adjuncts in certain forms of pyorrhea. The significance of the order in which the organisms are reduced may only be guessed at. It may be that the more resistant ones are more pathogenic. Again, it may be that, if the scaling of teeth is less effective in removing these organisms, something other than technical procedure may be more useful. These guesses suggest the importance of a type of "finding" which may be too summarily disregarded. Such data may later fit into a hypothesis like bricks in a building, and also serve to orient or suggest other problems. We must first get the facts before we theorize.

V. Studies in Bacteriology as Related to Formation of Calculus (Halituous fetor)

Further work was now projected in bacteriology. The clinical importance of calculus suggested the desirability of investigating the possible relationship of calcium-precipitating bacteria and mass reaction-changes in the saliva to pyorrhea. Ivan C. Hall obtained the following data:[9]

(1) A mass reaction-curve due to the effect of bacterial activity in saliva in vitro.

(2) Halituous fetor may, but does not necessarily, accompany salivary putrefaction.

(3) Halituous fetor is due to organisms destroyed by boiling, while putrefaction may be due in part to organisms not destroyed by boiling.

[9] Hall, Ivan C. Bacterial factors in pyorrhea alveolaris. I. Microbic changes in saliva. *Journal of the American Medical Association*, 1923, v. 81, pp. 1676–1679.

(4) Halituous fetor never occurs while fermentative changes are in progress.

(5) Squamous epithelial cells retard the reaction changes in collected saliva—presumably by their buffer action.

(6) Saliva, freed from epithelial cells by centrifugation, does not become fetid.

(7) The extent to which stagnation of proteins, including desquamated epithelium, occurs under the gingival margin may be important in reference to calculus formation. Note: This fits in with the importance of epithelial cells as matrix for calculus, as shown in figure 19, "Etiology of Parodontoclasia" (see footnote 4); also with observations of calculus formed from dead epithelial cells in the gums of an experimental subject fed for a week through a tube (to be reported later).

Dr. Hall, in a later publication, ascertained these additional facts:[10]

(1) The reaction curve for incubated saliva is characteristic, whether the samples are gathered from healthy mouths or mouths in which dental disease is present.

(2) This reaction curve is uninfluenced by differences in age and nationality.

(3) Close stoppering of incubated saliva prevents the alkaline change so characteristic of loosely stoppered or open samples, but does not prevent the development of fetor.

(4) Bacterial changes in stagnating saliva may influence the precipitation of dental calculus. But if alkaline changes were responsible for the precipitation of mineral salts in dental calculus, the fact that calculus is produced in the region of the gingival margin rather than in the depth of the pyorrheal pocket might be due to the release of CO_2 at the latter site.[11]

(5) Mineral salts are more abundant in the fluid of fresh saliva than in the cells.

[10] Hall, Ivan C., and Westbay, Clayton. Bacterial factors in pyorrhea alveolaris. II. Further studies on reaction changes in saliva, due to micro-organisms, and their possible relation to the formation of dental calculus. *Dental Cosmos*, 1925, v. 67, pp. 115–124.

[11] It is shown in fig. 15, "Etiology of Parodontoclasia" (footnote 4), that calculus can form to the depth of the pocket, which, however, is a field of necrotic tissue suitable to calcification.

(6) Although no significant variation in the total content of dissolved mineral salt could be detected after incubation for varying periods of time, either in closely stoppered samples whose reaction remained neutral or in loosely stoppered samples whose reaction became alkaline, yet calcium was strongly precipitated in each case in about forty-eight hours.

Clark and Levine[12] subsequently showed that removal of desquamated epithelial cells, food particles, etc., by centrifugation, results in loss of both calcium and phosphorus, probably as a calcium phosphorus mucin complex.

Work was undertaken by Loomis, under the direction of Dr. C. L. A. Schmidt, on mucin as a possibly important factor in the formation of calculus. Excellent progress was made, but since it was proved that a practical maturing of the experiments could not be expected within the time that remained during which support was assured, this work was discontinued for the time being.

It was pointed out by Professor Kofoid[13] that the association of oral amebae with filaments of leptothrix, and the secretion by the latter of salts of lime, is of considerable interest in reference to dental calculus; and that it should be noted, in this connection, that filamentous bacteria are associated with deposition of salts of manganese, iron, and calcium elsewhere in nature. The sagacity of this view has been confirmed by the recent brilliant work of Carl Naeslund.[14]

Finally, however, it should be borne in mind that salivary calculus is not necessarily related to disease. It is found in certain regions where oral stagnation occurs in animals having perfectly healthy mouths.

VI. Oral Anaerobes

Considerably more has been written on bacteriology in relation to pyorrhea than in any other province of biologic science. Yet important fields remained and still remain to be covered. One of the out-

[12] Clark, Guy W., and Levine, Lena. Inorganic constituents of human saliva, II. *American Journal of Physiology*, 1927, v. 81, pp. 264–275.

[13] Simonton, F. V. Progress report of the California Stomatological Research Group. *Journal of the American Dental Association*, 1926, v. 13, pp. 1586–1592.

[14] Naeslund, Carl. Studies of tartar formation. *Acta Path. et Microb. Scand.*, 1926, v. 3, pp. 637–677.

standing omissions was in the field of anaerobes. For this reason, and also because the bacteriologist in charge was particularly fitted for such an investigation, a study of the presence and possible rôle of anaerobic bacteria in dental infections was undertaken by Dr. Hall, with these results:[15]

(1) Sporulating anaerobic bacteria are only occasionally demonstrable in the saliva as spores.

(2) *B. welchii*, *B. tetani*, *B. bifermentans*, and *B. tetanomorphous* have been recovered from saliva.

(3) These organisms are evidently transient saprophytes in the mouth.

(4) Aerobic sporulating bacteria are evidently transient saprophytes in the mouth.

(5) There is no evidence that the sporulating aerobes or anaerobes have any direct pathological significance in relation to pyorrhea, but certain species may participate in the deposition of salivary calculus.

Later, the following additional facts were shown by Dr. Hall:[16]

(1) There occurs frequently in human saliva a minute gram-negative, non-sporulating, gas forming obligate anaerobe—the *Mic. gazogenes*. This organism has been overlooked in most of the modern work on the mouth flora.

(2) The above organism is non-pathogenic directly for rabbits, guinea pigs and mice, but appears to reduce the resistance of rabbits toward the pathogenic microbes of which they are carriers, notably coccidia.

(3) Normal rabbits, while they may harbor *Mic. gazogenes*, produce no agglutinins except under inoculation.

(4) Human beings not infrequently produce low grade agglutinating serums against *Mic. gazogenes*, the strongest serum coming from persons who are free or practically free from gingival lesions, suggesting an immunologic factor relating to this organism in pyorrhea.

It is interesting to note, apropos of the choice of this subject for

[15] Hall, Ivan C. Bacterial factors in pyorrhea alveolaris. III. The isolation of *B. tetani*, *B. welchii* and other sporulating anaerobes from human saliva. *Journal of Infectious Diseases*, 1925, v. 37, pp. 87–92.

[16] Hall, Ivan C., and Howitt, Beatrice. Bacterial factors in pyorrhea alveolaris. IV. Micrococcus gazogenes, a minute, gram-negative, non-sporulating anaerobe prevalent in human saliva. *Journal of Infectious Diseases*, 1925, v. 37, pp. 112–125.

study, that Ernest E. Glynn, Professor of Pathology, University of Liverpool, says (*British Dental Journal*, 1923, xliv, p. 682): "All the mouth anaerobes should be restudied with the same skill and care recently devoted to those from war wounds."

VII. Mineral Metabolism

Recognizing the importance of nutrition to the experimental production of dental disease, work was undertaken in this field. Since other workers had conducted, and were conducting, experiments in vitamine deficiency (in the laboratory of McCollum, for example) on a scale that we could not duplicate, and with a precision that did not require duplication; since the importance of mineral metabolism was evident; and since, furthermore, facilities and personnel for handling such a problem were available, it was undertaken to study, first, the mineral metabolism of adult man under varying dietetic conditions, and second, the inorganic constituents of the human saliva. Such was the program at the time a grant was made to the "Stomatological Group," as we called ourselves. Our problems had by this time grown to the point where support was of critical importance to continue them adequately. Professors L. B. Mendel and E. V. McCollum had visited San Quentin Prison and observed in particular the work in mineral metabolism. On Professor Mendel's return home he urged adequate support for this project. A considerable sum was necessary for the year. Consequently, the first-year grant was greedily absorbed in the work already projected and in certain further studies discussed below.

VIII. Original Orientation of the California Stomatological Research Group

The original idea in our group research was not alone or even primarily to study the treatment of pyorrhea, nor even to strive to ascertain the cause, but to gather together from as many fields as possible men who were thoroughly trained, and specialists in their branches, to study pyorrhea in a complete and scientific manner. The grant itself was made in broad terms for the study of pyorrhea and allied conditions—and for the continuation of an organization already

in operation. In spite of the broad terms of the grant, we felt that we should make our specific goal the etiology of pyorrhea—treatment was and has been an ancillary.

We have found it necessary, both for scientific and practical studies, to work out very accurate diagnostic methods, and these have been proven necessary and invaluable in the study of treatment. Treatment, however, is primarily a study for the dentist and clinician. We have felt that specialists in science could best do, and should do, work which cannot be done by the dentist and clinician.

Our first orientation, in spite of a specific goal, was to deploy in search for clues. In our later work we aimed to narrow down and follow the most promising clues. This we have done.

IX. Early Projected Studies

In addition to the work originally under way, we projected these studies:

Bacteriology. A study of the possible correlation of the bacteriophage with clinical dental conditions.

Histopathology. (1) An endeavor to reproduce in dogs, rabbits, and guinea pigs, a lesion similar to pyorrhea in man. Injections of fresh pyorrheal exudate to be made deep into the periodontal membrane. In other animals, irritation to be produced by placing inert finely divided materials in contact with the periodontal membrane.

(2) A series of experiments on dogs, rabbits, and guinea-pigs, to observe how repair takes place following trauma to the alveolar process, pericementum, cementum, and dentine.

(3) A study of the relation of clasmatocytes to pyorrheal conditions.

(4) Studies of human biopsy material from cases of pyorrhea.

Protein chemistry. In order to profit by the coöperation of the head of the Biochemistry Department, an expert in protein chemistry, it was planned to study the organic constituents of saliva, beginning with mucin as a possible factor in the formation of calculus.

Endocrinology. Work was contemplated in this field, if a competent investigator could be secured.

Protozoology. Although it seemed probable that mouth protozoons were only of ancillary importance in pyorrhea, it was nevertheless

decided to allot limited support in this field because of the eminence of the scientist who would have the work in charge. Subsequent results have more than justified this decision, since an abundance of work of great scientific value has resulted.

Focal infection. Since unusually favorable clinical opportunities were available, work was undertaken in this as an ancillary field.

Clinical dental studies. Studies were undertaken in diagnosis of degree of pyorrheal involvement and in effectiveness of treatment by the conservative method; and the same plus endocrine treatment, therapy by cod liver oil and calcium lactate, and local germicides.

While these projects were maturing, watch was kept as to their promise of contributing to our knowledge of pyorrhea and its cause. During the earlier years, studies were always completed. Later, however, it was felt necessary to leave unfinished some very promising scientific studies which did not show promise of contributing specifically to the knowledge of pyorrhea or of maturing within the period of our grant. Realizing always the importance of nutrition, the Group, during the conduct of the chemical studies in mineral metabolism, extended its activities into the field of animal experimentation. At the time when Professor Gies suggested the publication of a general report of our findings,[17] we had approached the end of our third year under the Carnegie grant. In addition to the findings already mentioned above were the following in that report:

Bacteriology. Items 23, 24, 25, 26,[18] 31, 32, 33, 34, 35[19]

Protozoology. Items 36 to 56 inclusive

Histopathology. Items 57 to 68 inclusive

Nutrition. Items 68 to 82 inclusive

Dental studies. Items 83 to 91 inclusive

Medical studies. Items 93 to 102 inclusive

During this time our orientation was studied. Those fields which seemed most profitable and apropos were strengthened and others discontinued, or placed under different auspices, as follows:

It became apparent that experiments on diet, with clinical and

[17] Simonton, F. V. A report on some of the findings of the California Stomatological Research Group. *Journal of Dental Research*, 1927, v. 7, pp. 239–44.

[18] Practical application of this work has been made in the preparation and use of antogenous vaccines.

[19] A study of the therapeutic value of instillations of bacteriophage is contemplated.

histopathologic study or both, is the most immediately fruitful field in the study of the etiology of pyorrhea.

The chemical studies in mineral metabolism were not showing any significant correlation between the presence of Ca, Mg, Na, Cl, P, etc., in the food ingested and their reappearance in blood, urine, feces, and saliva. Hence the original plan to continue this study on pyorrhetics was abandoned.

This and subsequent experience has convinced us that the time has not yet arrived to mount the crest of the wave in application of laboratory chemical technic to pyorrhea. Nevertheless, a monumental scientific contribution was made under unique conditions, the ultimate value of which can not be foretold, but which will serve as a stepping stone when the time comes for further application of such methods.

The work in animal experimentation, items 72 to 82, inclusive, of the report (see foot note 17), showed great promise. It was continued, and a parallel project gotten under way to test on men at San Quentin the results secured in animals.

In our work on this problem at San Quentin, we found ourselves confronted with the necessity of perfecting methods of clinical diagnosis. This proved to be a complicated but very instructive problem. The chemist wished us to select three groups—healthy, medium pyorrhea, and advanced pyorrhea. This required superficial examination of hundreds of men to find the requisite number of the same race, age grouping, free from systemic illness, willing, sufficiently intelligent, and with sentences long enough to ensure their stay during the experimental period. We selected our groups on the basis of clinical symptomatology, and then proceeded to make full mouth radiographs. We had recognized two types of pyorrhea—the inflammatory and the non-inflammatory—but nevertheless the observation from the radiographs, which clinical symptoms did not disclose or parallel bone wastage, was impressive. This meant a re-evaluation of our cases.

Since the most significant results in animal experimentation had to do with the non-inflammatory type, we selected cases where the bone atrophy was more marked in degree than the inflammation, and arbitrarily classified them, giving the greatest weight to bone atrophy, next to pockets, and last to filth. The men were placed in three

groups (immune, medium pyorrhea, and advanced pyorrhea) on high protein acid diet, and in three groups on high carbohydrate basic diet. Their mouths were examined with the utmost care. Detachments were measured according to methods described in our publications. Standardized x-rays were taken with the device recently described.[20] Careful observation and record of gingival stigmata were made according to a method described in the conclusion of the discussion of the present paper (page 270).

In a year's time, on diets as acid or as alkaline as they could be made on natural foods, our cases failed to reveal any significant change detectable by the most accurate methods we were able to devise. I recall a case that I followed a year without being able to detect any change. The patient was then removed from the diet squad as a result of having contracted tuberculosis. A month later he had developed a granulation septum of 3 mm. between a lower left lateral incisor and cuspid. The only change in his diet in the tuberculosis ward was an increase in eggs and milk. The negative findings on the men do not invalidate the significance of the experimental production of non-inflammatory pyorrhea in the dogs.

This work on both animals and men was reported by Dr. Martha R. Jones at the last meeting of the Chicago Dental Society (1927). A critical survey was made by a consulting nutrition expert of all the evidence in this research, and he evaluated the argument for the experimental production of non-inflammatory pyorrhea as cogent. What the negative findings on the men at San Quentin does show is that changes occur too slowly in adults to allow detection within the limits of time on which it is practical to restrict them to an experimental diet. If tissue could be excised *ad lib.* and studied microscopically we could doubtless detect changes. All this meant re-orientation into animal experimentation where we could remove tissue when necessary.

X. Histopathology

Much material was prepared and studied as a basis for the report given by Dr. Jones in Chicago. Studies were also made of the teeth

[20] Simonton, F. V. Precision device for duplicating roentgenographs. *Dental Cosmos*, 1928, v. 70, pp. 13–15.

and investing tissues of coyotes, monkeys, wildcats, and opossums. A wealth of material has been accumulated and is being studied. With full realization of the importance of this field we have been working more toward the development of an organization of this work rather than trying for hasty results. Two years elapsed before we had developed a histologic technique which was satisfactory.[21]

The development of dental histopathology in this country has lagged far behind that in Austria and Germany. These countries have a leadership in microscopic technique and research comparable to the ascendancy of Italy in painting and music, and ancient Greece in sculpture.

Two more years elapsed before we could secure a suitable scholar from Germany to undertake this work. During the interim we carried on in this field with the facilities and personnel available. We now have a well-trained investigator who has organized an adequate department and who has been provided with a wealth of material.

XI. Further Studies in Biochemistry

In a paper on the influence of diet upon the inorganic constituents of human saliva,[22] the authors presented these conclusions:

"From the results of a series of diet experiments with five human subjects, in which the ingestion, excretion and retention of calcium, magnesium, sodium, potassium, chlorin, phosphorus, sulphur and nitrogen are definitely known for a period of twenty-eight weeks, it is evident that:

"(1) Because of previous ingestion of inadequate diets, our subjects were in great need of the various inorganic elements. During the entire experimental period these men retained large amounts of base, varying from 64 to 227 cc. of tenth normal solution per day.

"(2) Within reasonable limits the acid-base balance of the foods ingested has little or no influence upon the ability of the adult to retain base. During the experimental period, the weekly acid-base balance of the foods varied from 3.5 cc. of normal acid to 480 cc. of normal base and yet with but one exception all subjects showed base retention.

[21] Williams, Adrienne. The preparation of combined hard and soft tissues for histological study. *Dental Cosmos*, 1927, v. 69, pp. 715–721.

[22] Clark, Guy W., and Shell, John S. The influence of diet upon the inorganic constituents of human saliva. *Dental Cosmos*, 1927, v. 69, pp. 500–513.

"(3) A diet containing 'green foods' (potatoes, peas, string beans) increases the retention of calcium and phosphorus. This diet also has a beneficial effect on general nutrition.

"(4) It has not been possible to demonstrate the existence of relationships between the amount of a given element ingested or retained and the amount appearing in the circulating blood or in the resting saliva.

"(5) Although the constituents appearing in the saliva must primarily be supplied by the circulating blood (and lymph), it has not been possible to show any relationship between the concentration of the inorganic constituents of the blood and the amount appearing in the saliva.

"(6) The saliva contains relatively large amounts of inorganic phosphorus, from three to five times as much as is found in blood plasma. This inorganic phosphorus is important because it provides the saliva with an excellent buffer system."

In a second study, on the inorganic constituents of human saliva, these conclusions were published:

"(1) The inorganic constituents of paraffin-activated saliva show greater quantitative variations than those of resting saliva.

"(2) Removal of desquamated cells, food particles, etc., by centrifuging, results in a loss of both calcium and phosphorus. It is suggested that these elements are removed in the form of a Ca-P-mucin complex rather than as $Ca_3(PO_4)_2$ or $CaHPO_4$.

"(3) It is not possible to appreciably change the composition of the saliva by short fasting.

"(4) It is difficult to increase the chlorid content of resting saliva by the oral ingestion of large amounts of soluble chlorids.

"(5) With respect to abnormal ingestion of inorganic phosphorus the salivary glands exhibit a marked excretory function.

"(6) Variations of the several constituents of the saliva may be the result of physiological rhythm.

"In conclusion it may be said that each individual reacts differently to stimulation, and the individual response to the same type of stimulation varies from time to time, with the result that the normal variations in the content of the respective inorganic constituents are sufficiently large to disguise any relationship that may exist. Our results are therefore in accord with the earlier work of Chittenden and Richards, Lothrop and Gies, Spencer-Payne, and with that reported in a previous paper."

In a study on calcium distribution in blood serum,[23] the authors stated:

"(1) The method devised by Moritz for the determination of diffusible calcium in blood serum has been improved in a number of respects.

"(2) A study has been made of the distribution of diffusible and non-diffusible calcium in the blood serum of eighteen normal young men and women, two rabbits, two dogs, and two calves.

"(3) Attention is called to an error made by Moritz in the calculation of diffusible calcium in the blood serum of rabbits and, by applying a probable correction, the figures given by him for normal rabbits have been recalculated.

"(4) The results on the serum of human subjects and the recalculated results of Moritz on the serum of rabbits indicate that the concentration of diffusible calcium tends to remain more constant than the concentration of the non-diffusible form.

"(5) It has been observed that the non-diffusible calcium is not in equilibrium with the total diffusible calcium, although it seems probable that it may be in equilibrium with a small fraction of the total diffusible calcium present in blood serum.

"(6) Evidence has been brought forward which seems to indicate that the non-diffusible calcium is bound to the serum proteins or some component of the serum proteins in a non-ionized form."

Results of experiments on the reaction changes of human saliva[24] were reported in these conclusions:

"(1) The saliva obtained by direct cannulation of the human parotid and sublingual glands is but slightly more acid, 0.1 pH, than freshly expectorated resting saliva.

"(2) Although the volume per cent of carbon dioxide is much higher in paraffin-activated saliva, it varies in much the same manner as in resting saliva.

"(3) Samples of either resting or paraffin-activated saliva may stand for several hours without showing appreciable changes in the carbon dioxide content. This is explained by an equilibrium between the carbon dioxide escaping and that being formed.

[23] Updegraff, Helen, Greenberg, David M., and Clark, Guy W. A study of the distribution of the diffusible and non-diffusible calcium in the blood sera of normal animals. *Journal of Biological Chemistry*, 1926, v. 71, pp. 87–117.

[24] Clark, Guy W., and Carter, Kenneth L. Factors involved in the reaction changes of human saliva. *Journal of Biological Chemistry*, 1927, v. 73, pp. 391–404.

"(4) The formation of carbon dioxide is probably the result of enzymatic action.

"(5) There is apparently some ammonia formed by bacterial action. It is thought, however, that most of it is the result of enzymatic action.

"(6) There is no demonstrable relationship between the pH, the volume per cent of carbon dioxide, and the ammonia content. The pH changes in saliva apparently involve other constituents than those studied. To complete this aspect of the work properly it will be necessary to make complete analyses of a large number of samples of saliva."

XII. Further Studies in Nutrition

In studies on inorganic salt metabolism in dogs, these findings were reported:[25]

"(1) Normal puppies kept in indoor pens grew satisfactorily and showed no evidence of faulty mineral metabolism on diets consisting of certain fixed quantities of meat, milk, butter fat and orange juice, supplemented with a sufficient quantity of wheat bread to meet the caloric requirements of the growing animal. Puppies with an inherent metabolic fault developed more or less marked rickets-like changes on the same diet under identical conditions.

"(2) The retention of calcium and phosphorus by normal puppies on the basal diet with the addition of a phosphorus-free salt mixture high in calcium and low in potential alkalinity; and the same, supplemented with sufficient dibasic potassium phosphate to make the phosphorus content relatively high, were of the same order of magnitude. Skeletal development appeared to be normal in all cases. Subnormal puppies showed profound retrograde bone changes on the same diets and under identical conditions.

"(3) Normal animals tolerate well a certain excess of alkali in diets both high and low in phosphorus, but if the amount is sufficiently increased, the calcifying mechanism is reversed. Neutralization of the excess of alkali initiates the healing process, which progresses rapidly in animals in which retrograde bone changes have been induced and are not too advanced, and more slowly in those animals with an inherent metabolic fault.

"(4) The calcium-phosphorus concentration product of the blood plasma was found to be well within the normal range in young puppies in the early

[25] Jones, Martha R. Studies on inorganic salt metabolism in dogs. III. On certain factors which influence the deposition and resorption of bone. *American Journal of Physiology*, 1927, v. 79, pp. 694–707.

stages of rickets. In more advanced cases in older animals the phosphorus content was markedly reduced.

"(5) Metabolism and blood studies indicate that the seat of the disturbance in faulty mineral metabolism is in the tissues rather than the gastrointestinal tract.

"(6) It appears that calcification is dependent upon two factors—one, and the more important, being the metabolic state which is inherent in the individual, and the other—diet, which plays an important part in regulating cell metabolism. It is believed that the precipitation of bone salts is determined by the balance of ions between the bone-forming cells and their surrounding medium, and that any agent (diet, hygiene, ultraviolet light, active principle in cod liver oil, etc.) which either directly or by catalysis influences the metabolism of the bone cells will alter that balance and thus become an important factor in regulating the deposition and resorption of bone."

In reporting on dental caries and pulp sequelae resulting from experimental diets, the author stated:[36]

"(1) Caries has not been found in young animals, even those of a third generation, from parents on a diet deficient in vitamin A. After maturity has been reached, caries develops.

"(2) The experimentally produced caries is more similar to that found in adult man than in the child or adolescent.

"(3) Pulp lesions, such as pulp abscess and pulp calcification, are demonstrable in teeth that do not show caries as well as in those that do.

"(4) The average growth curves for all the experimental animals indicate typical vitamin A avitaminosis.

"(5) All the animals do not respond to the same degree to the deficient diets.

"(6) Without regard to growth weights, rats, both male and female, on vitamin-A-deficient diets, develop dental caries after maturity has been reached.

"(7) Up to this time at least, the dental defects observed in dogs on deficient diets are not comparable to those reported on rats, for neither enamel nor dentin hypoplasia have been found.

"(8) A relatively high degree of attrition and abrasion of tooth surface occurs both in normal rats and in those fed on diets deficient in vitamin A, a

[36] Marshall, John Albert. Dental caries and pulp sequelae resulting from experimental diets. *Journal of the American Dental Association*, 1927, v. 14, pp. 3–37.

condition that may or may not have some bearing on the development of carious processes in the second group of animals.

"(9) Up to the present, it has not been found possible to alter or arrest caries when an animal previously on a vitamin-A-deficient diet is fed vitamin A at a later period."

XIII. Further Studies in Protozoology

A study of the cultivation of *Trichomonas buccalis*[27] afforded the results in this summary:

"(1) Several culture media have been devised which give excellent growth of *Trichomonas in vitro*.

"(2) *T. buccalis* will survive a temperature of 0°C. for 48 hours; 10°C. for 72 hours; room temperature for 3–6 days; 45°C. for 9–11 minutes; and 47.5°C. for 7–9 minutes.

"(3) *T. buccalis* will not survive complete desiccation in dilute aqueous solutions.

"(4) This organism will withstand suspension in distilled water for 10–12 hours.

"(5) In the presence of air *T. buccalis* is killed in less than 7 minutes upon exposure to a Kromayer water-cooled quartz mercury arc lamp. In the absence of free oxygen this treatment may be continued for 1–2 hours.

"(6) *Trichomonas buccalis* may multiply for 72 hours in contact with a capsule containing 50 mg. of radium.

"(7) Oxygen is toxic for *Trichomonas buccalis in vitro*. All parasites directly exposed to the action of the gas are usually killed by 30 minutes of slow bubbling of this gas through the cultures. Forms hidden in debris may escape this lethal action of the gas. This parasite is probably an obligate anaerobe.

"(8) The *Trichomonas* of the mouth multiplies in a relatively wide range of hydrogen ion concentrations. The activities of the bacteria in the cultures so mask the activities of the protozoan as to make biochemical studies of the latter impossible.

"(9) The split products of a number of proteins may be utilized by *T. buccalis*. The simplest medium yielding growth was pancreatic digest of sodium caseinate highly diluted with Locke's solution, forming a 0.5–1 per cent solution.

"(10) Carbohydrates may be omitted from the formulas for culture

[27] Hinshaw, H. Corwin. Cultivation of *Trichomonas buccalis*, a protozoan of the human mouth. *University of California Publications in Zoology*, 1927, v. 31, pp. 31–51.

media without destroying their power to support the growth of this parasite. In the absence of this carbohydrate the reaction of the medium becomes alkaline very quickly.

"(11) We have not succeeded in cultivating *T. buccalis* free from bacteria although the isolation of this protozoan is not impossible.

"(12) Contamination of cultures by certain bacteria has resulted in a great acceleration of growth. Many other bacteria are decidedly antagonistic to this flagellate *in vitro*.

"(13) *Trichomonas buccalis* can be grown by hanging-drop tissue-cultures of chick-embryo liver with bacteria. In the absence of bacteria, radical autotomy and death ensue within two hours."

XIV. Further Studies in Bacteriology

A study of effects of various diets upon the hygiene and microbiology of the mouth, without and with the use of the tooth brush,[28] was made; also a quantitative examination of the mouth flora under different dietary conditions.[29]

XV. Present Activities

Work this year is concentrated upon experiments with rats, dogs, and monkeys. Those clinical data are being collected which appear *a priori* relevant, or which experience has indicated as relevant. Particular emphasis has been placed upon microscopic study of biopsy and post-mortem material. Through the co-operation of Professor K.F. Meyer, Director of the Hooper Foundation for Medical Research, a most modern and complete laboratory has been provided for pathological research and placed under competent direction.

If future resources permit a continuation of the activities of the Group for a reasonable period of time, the outlook for further important contributions is very promising.

[At the close numerous illustrations were projected on a screen and briefly discussed. None of these have been submitted for publication here, all of them having been included in papers named in the footnotes.—(*Ed.*)]

[28] Howitt, Beatrice F., Fleming, Willard C., and Simonton, F. V. In press—*Dental Cosmos*.

[29] Howitt, Beatrice F., and Fleming, W. C. In press—*Journal of Dental Research*.

XVI. Discussion

A. Introduction

Leuman M. Waugh, D.D.S., F.A.C.D., Chairman (New York City), representing the New York Section of the International Association for Dental Research: In the absence of the Chairman of the New York Section of the Research Association, it becomes my pleasant duty, on behalf of the section, to welcome you to this gathering. We deeply regret that our Chairman, Dr. J. Leon Williams, is incapacitated and unable to be with us. He asked that his greetings be extended to all in attendance.

Some of the members have asked just how it happened that we are having a joint meeting this evening. I might say, by way of explanation, that the New York Section of the International Association for Dental Research had planned a program of five papers at a meeting called for tomorrow evening. But when we learned that so rare a treat as Dr. Simonton's report might be brought to New York within a day or two of that time, I had the courage, after conferring with some of our officers, to ask our members on the program to permit the postponement of that program until our next meeting, in January or February, to enable us to enjoy Dr. Simonton's report, which suggestion was eagerly accepted. Then it seemed particularly opportune to have the meeting in conjunction with the New York Academy of Dentistry, which had not scheduled a meeting for December, and to open the meeting to all who might wish to come. The attendance, it would seem, bespeaks the sanction of your officers' action.

By a happy coincidence, the man who had been selected by Dr. Williams, to occupy the chair at the next meeting of the New York Section of the Research Association, is also President of the New York Academy of Dentistry. I take great pleasure in introducing the President of the Academy, who will preside at this meeting: Dr. Merritt.

President Arthur H. Merritt, D.D.S., F.A.C.D., F.A.A.P. (New York City): As President of the New York Academy of Dentistry, it affords me great pleasure to extend a very cordial welcome to Drs. Duel and Semken, representatives of the medical profession of New York; Dr. Thoma of Boston; Dr. Rickert of Ann Arbor; Dr. Blumenthal of Boston; Dr. Kelsey of Baltimore; Dr. Hinman of Atlanta; Dr. Detlefsen of the Wistar Institute, Philadelphia; Dr. Appleton of the University of Pennsylvania; Drs. Keppel and Furst of the Carnegie Corporation; and all other visiting colleagues, whose presence here tonight is very agreeable to us.

Dr. Waugh has explained to you the nature of this meeting. As you

know, Dr. Simonton has been engaged for several years in an investigation of problems in periodontia. His work interests not only the periodontist but every one engaged in the prevention and treatment of disease.

[The paper on pages 235–258 was then presented by Dr. Simonton, who revised it for publication, and corrected the proof.—(*Ed.*)]

B. CONCLUSION

President Merritt (*New York City*): It is manifestly impossible for me, as a clinician, adequately to discuss the subject so ably presented by Dr. Simonton, without having had an opportunity to study it—which I have not had. I have marvelled at the amount of ground covered by Dr. Simonton, and his familiarity with his subject. I have been wondering how it was that one mind could comprehend so much, and how a mere clinician could be expected to discuss a subject so comprehensive.

Diseases of the periodontium are as old as civilization itself, perhaps older. They seem always to have been shrouded in mystery. Fifteen hundred years B.C., the Chinese were studying these same problems which we are considering tonight. A favorite remedy at that time was the use of human urine. In 1728, when Fauchard published his epoch-making book on dentistry, in which he devoted many pages to a description of pyorrhea, he, too, was recommending urine as a mouth wash. In this period of more than three thousand years, about the only progress that seems to have been made in treatment was the recommendation that one should use his own, and not his neighbor's urine. In 1928, two hundred years later, we are no longer using urine in treatment. Instead, we use "Mercitan," "Pyorrhicide," and "Forhan's for the Gums." These may be pleasanter to use, but whether they are any more efficient is an open question. In one respect, at least, the older remedy had something to recommend it—it was not sold to the public at a dollar per ounce.

I am sorry that Dr. Simonton should have felt it necessary to create a new nomenclature. If there is any one thing more than another that has set back the wheel of progress, it has been confusion in nomenclature. One hundred years ago "Riggs' disease" was the term used to cover the group of diseases under consideration tonight. This was followed by "Pyorrhoea alveolaris," "Interstitial gingivitis." Later, under the direction of the American Academy of Periodontology, the term "Periodontoclasia" was given to it. Now we have also such terms as "paradontoclasia," "parodontitis," "parodontoses," etc., until it has become a case of "confusion worse confounded." This is unfortunate.

These researches, so ably carried out by Dr. Simonton and his co-workers,

are most interesting and no doubt valuable. The test of their value, however, is to be found in their clinical application. No matter how interesting they may be of themselves, unless they contribute to our knowledge and control of the diseases which involve and break down the supporting tissues of the teeth, they will have failed of their purpose. What the clinician asks of the research worker is: What have you to offer that will make it possible for the man who stands on the firing line of daily practice to render a more efficient service? This is the question which I would ask of Dr. Simonton tonight, and I hope he will, in closing the discussion, point the clinical application of his most interesting and comprehensive report.

Isador Hirschfeld, D.D.S. (*New York City*): I feel it a great honor to have been asked to contribute to the discussion of so interesting a lecture. It is very evident that the lecturer knows his subject well, and that he can convey his knowledge clearly to others. I was certainly enchanted with many of the things that I heard, some for the first time and others that I had previously read. It was most interesting to me who, at best, can afford to spend comparatively few hours a week, outside of the office, in experimentation and investigation in hospitals and dispensaries, along clinical lines. I understand that it is from this clinical view-point that I am expected to discuss Dr. Simonton's presentation.

Most gratifying is the fact that Dr. Simonton and his collaborators make it a point to record clinical pictures of the parts they describe histopathologically. This helps considerably to stabilize research, and we would all like to see more of this high-grade work. It is very disappointing to read histopathological reports without getting, at the same time, the complete clinical picture and history of the individual cases, thus often losing the important link in the chain.

Regarding clinical reattachment: I believe it occurs. The first of the experiences that convinced me of this was an instance of a pocket in which the apoxesis had not been done completely—I had overlooked and failed to remove some material at the very bottom of it. The pocket became gradually narrower. Later, on reinspection, I inserted a very fine instrument in this formerly broad pocket but could not move it laterally—the reattachment seemed to grow in from the sides. An instrument might be prevented from getting down into a pocket because the gingival tissue contracted sufficiently to hug the root of the tooth tightly; but if the instrument could go down, as in this case, yet could not be moved from side to side, it is logical to think there was lateral attachment. On the other hand, in cases successfully treated, very frequently even fine wires cannot

be run down without pain and hemorrhage into pockets that at one time were very deep. This, I should think, would indicate absolute reattachment. It was only last week that I again made such an observation when I x-rayed a tooth for checking up purposes. Six years ago, when I first treated that tooth, it had a very deep pocket extending almost to the apex (*fig. 1*). The patient, because of her own curiosity and because she thought I might be interested at this time in seeing the progress of the tooth, came to the office to have me look at it. I found a very nice regeneration of bone (*fig. 2*). I tried to run a fine instrument into the pocket, but could not

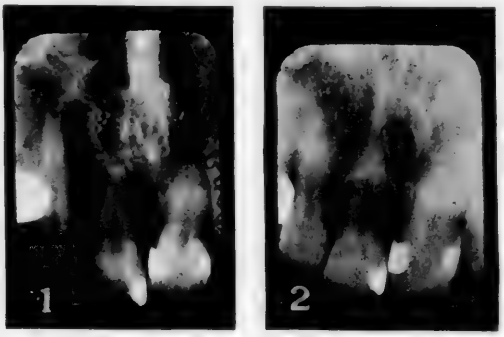


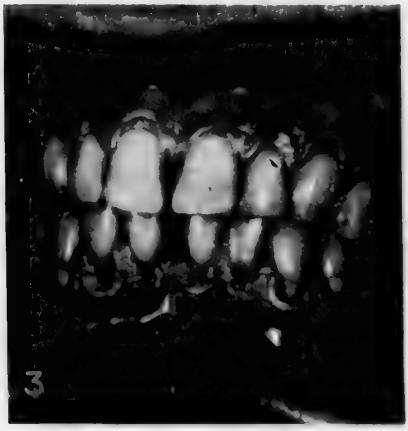
Fig. 1. Upper central incisor, elongated and pushed out of alignment labially by an inpacted cuspid, which had recently been extracted. Marked alveolar destruction can be seen all around this tooth. There were deep pyorrheal pockets on the labial and palatal sides.

Fig. 2. Same as *fig 1*, six years later, showing filling in of bone and return of the tooth to its normal position in the arch without orthodontic aid.

possibly get beyond what we would probably consider the average normal gingival crevice. Neither could a fine diagnostic wire go up any further.

As to diet in relation to treatment of pyorrhea: I believe that dentists, not unlike physicians, in considering treatment of disease, should depend upon all factors that collectively favor the building up of a greater resistance, and diet is an important consideration. Nevertheless, it is true that many of us have had a very large percentage of what we believe really good results in the treatment of suppurative periodontoclasia without laying special emphasis on diet. I use the term "suppurative" in contrast to what the lecturer referred to as "dry pyorrhea." I do not know whether I understood him correctly or not, but it seems to me that what he alluded to as "dry" pyorrhea was probably not a pyorrhea at all. The additional

slides I am about to present will in part probably help to explain what I mean. Briefly, they show a type of case in which there is appreciable or



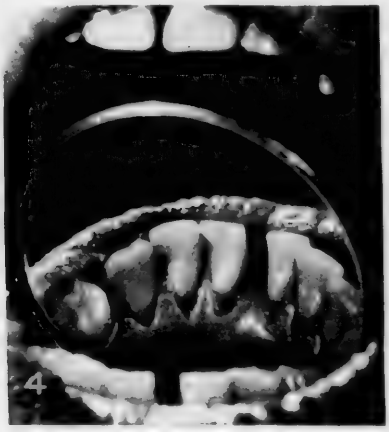


Fig. 3. Atrophic condition, showing a marginal recession. See *fig 4*.
Fig. 4. Same as *fig 3;* lingual view.

Fig. 5. In spite of the marked recession, both lingually and labially, all over the mouth, only three pyorrheal pockets were found—on the labial side only of the lower right lateral incisor and cuspid, and of the lower left central incisor. In this mouth, the buccal bifurcations are exposed, but the gingival margin around the individual root is not detached, and no pus is present.

marked generalized periodontal destruction, some of which, because of the absence of pus, are frequently referred to as "dry pyorrhea." Upon close inspection, we find that macroscopic pus formation is not a "constant" in

the picture. In other words: in some of these, no pus at all may be found; in some, pus may be found at a few points of recession, but not at others; while in other instances, suppuration is evident at all points of periodontal

Fig. 6. Radiographs of the case illustrated in *fig. 5*

recession. All of the cases that follow give histories of faulty endocrine functioning coincident with symmetrically generalized bone resorption. This coincidence seems to point to a possible relationship between them, perhaps by way of faulty calcium metabolism, which may be worthy of serious consideration.

Fig. 3. A very interesting atrophic condition, showing a marginal recession. The synthetic fillings in the exposed roots were inserted six years

Fig. 7. Note the remarkably even recession all around the mouth

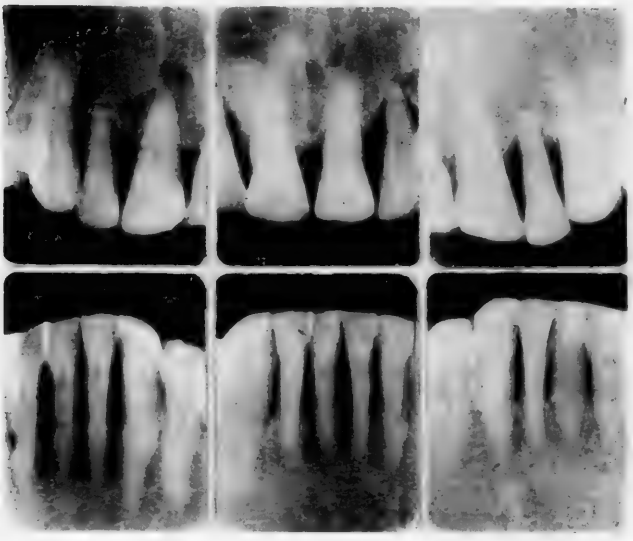


Fig. 8, A. Radiographs of the case illustrated in *fig. 7*

before the picture was taken. In the kind of dental work evident elsewhere in the patient's mouth, we could not expect a synthetic filling to last very long unless the cavity as well as the filling material had been kept perfectly

dry for a sufficient time—practically an impossibility any considerable distance underneath the gums. The perfect condition of these fillings and the position of the gingival margins right up to them indicate, therefore, that the recession had ceased at least six years before her visit to the clinic.

Fig. 8, B. Radiographs of the case illustrated in *fig. 7*

It has ceased without any special help, according to the patient's history. Her diet had not changed materially. See *fig. 4.*

Fig. 4. (The mouth shown in *fig. 3.*) A similar recession on the lingual side of the mandible. There is no salivary calculus. This girl gives a history of thyroidectomy. The fact that the progress of the recession was

checked automatically, that is, without any local treatment and without any decided change in nutrition, led me to think that the endocrine condition might have a definite relation to this particular condition. We know that all the endocrine glands may, at times, return to normal function when

Fig. 9. Before treatment. See *fig. 10*

Fig. 10. Same as *fig. 9;* after treatment. Since completion of the treatment, the teeth have been slowly moving back toward the arch. The comparison between the original cast and the one taken last shows a considerable decrease in the diameters of the dental arch.

the one which was perhaps responsible for the disturbance in their entire chain has been corrected.

Figs. 5 and 6. A symmetrically generalized advanced recession of all the gingivae. The patient came to me a month ago, and my first impression was that he had an advanced pyorrheal condition. To my surprise, however, I found that only a few of the lower teeth had suppurative pockets.

These were fairly deep and of the vertical type. Around the other teeth, the pockets were no deeper than the average gingival crevice and contained no pus. Evidently, the generalized great loss of the supporting structures was due to some cause other than infection, and the pyorrheal part was only a mild contribution to the picture as a whole. The patient is quite ill. From what his physician told him, he believes that he suffers from a pituitary disturbance. Radiographs are shown in *fig 6*.

Figs. 7 and 8. Photograph (7) recently taken of a patient treated eight years ago for a generalized periclasia. The perfect symmetry of the alveolar destruction was probably due to the fact that the teeth were evenly set. Had any of them been in improper coördination because of malposition, the destruction around such teeth would very likely have been much greater. (I have observed this fact in numerous cases of this type). He had pyorrhea in only some of the teeth. That the recession had ceased four years ago is now evident from a comparison with the casts made of his mouth at that time. This patient evidently was also an endocrine case, as he has recently had a goiter removed. Radiographs are shown in *fig. 8*.

Figs. 9 and 10. Girl of nineteen whom I commenced to treat about a year ago. She had a very marked destruction, and showed well established symptoms of endocrine disturbances. I therefore sent her to an endocrinologist. After an x-ray examination, he diagnosed the condition as that of either an abnormal or pathologic pituitary gland. He suggested thyroid therapy and she is now being closely observed from this angle. Her entire physical condition has improved remarkably and the periodontal disease readily responded to treatment.

J. Oppie McCall, D.D.S., F.A.C.D., F.A.A.P. (New York City): The first thing I would like to say relates to the matter of reattachment. I got from Dr. Gottlieb a little different version of his interpretation of Box's study of the tooth which Dr. Simonton mentioned than that given you tonight by Dr. Simonton. Dr. Gottlieb did take exception to Dr. Box's findings, or rather to his interpretation of those findings. He disagreed with nearly everything that Box said except with regard to the reattachment itself. He told me that he had no doubt that there had been reattachment, but he did not believe that it had been proved by the things Dr. Box pointed out. He mentioned more specifically than anything else the matter of formation of new cementum. In going over the specimen since then, I have become convinced that Dr. Gottlieb was right regarding that particular part of the specimen. But as regards the attachment of the epithelium, I think Dr. Gottlieb admitted that it had occurred; that the cementum had

been scraped with an instrument and was not eroded by any pathologic process. Dr. Simonton did not mention tonight the fact that between the points of epithelial attachment and where the new cementum showed, there was a considerable area of root surface having a connective tissue attachment. The parallelism of fibers to the root surface might also be shown in specimens such as Dr. Simonton put on the screen. Nevertheless, the evidence of the cementum surface itself demonstrated that there had actually been a pathologic detachment, and a subsequent reattachment of the tissue to that tooth.

Of course, clinically many of us are satisfied about the matter of reattachment. That particular point, however, does not come into the problem which we are discussing tonight. Dr. Simonton has told us that he is here to talk about etiology primarily, and has detailed the very numerous and interesting experiments which have been carried on under his direction in the attempt to establish etiology. I would like to call your attention to just one thing. I myself have given considerable attention to the etiology of this disease in the past, and I have made a statement with which other clinicians have agreed: "the etiology of periodontoclasia is very complex." We are very fond of talking about traumatic occlusion as a causative agent in periodontoclasia. No one, I believe, not even Dr. Stillman, who was the father of traumatic occlusion, has ever claimed that traumatic occlusion alone produces pyorrhea. We have always realized that it required the combined influence of several factors, of which traumatic occlusion was only one. We also recognize many cases in which suppurative lesions develop, but in which there is no traumatic occlusion—no occlusion at all. It is a complex thing.

I would submit that experiments to determine etiology must be conducted on the basis of a complex etiology. I do not believe that we are going to produce pyorrhea in anything like a typical form in a human being or in an animal by attempting to do just one thing to that animal and nothing more. I believe we must combine our different factors, starting preferably with those about which we already know and which we are able to demonstrate clinically, and then proceeding into the more obscure fields in which we are all interested and whose secrets we find so elusive.

Charles F. Bödecker, D.D.S., F.A.C.D. (*New York City*): It is needless to express my profound admiration for this work, for that has been evinced from all sides. I can well judge the amount of patience, preparation, and research it takes to perfect work like that which Dr. Simonton and his group have shown us, of which we have only seen the high spots at this meeting.

I desire to emphasize one point Dr. Simonton brought out; namely, the cause of many cases of periclasia is lack of cleanliness (Gottlieb calls it "Schmutzpyorrhoea"). The symptoms of this disease disappear rapidly under proper treatment, including thorough massage of the gingiva by the patient.

Dr. Simonton and his group undoubtedly attacked the problem in a fundamentally scientific manner, in attempting to find first the etiology. After the cause is found, then only can we hope to devise a cure.

President Merritt: In view of the fact that it is so late, we will close the discussion at this point, unless some one has something special he would like to say or ask. And I may say to Dr. Simonton that the questions that have been raised were asked because he seems to know everything about it, and we who have raised those questions know very little. We are very glad, Dr. Simonton, that you do know so much, and have come here to tell us about it. Now, we shall be glad to hear your remarks in closing the discussion.

Dr. Simonton (in conclusion): With reference to the question of terminology, I may say that we have not been concerned with nomenclature *per se*, but have nevertheless found it necessary to classify and delimit clinical entities in order that our associates in the biologic sciences could segregate their data.

I have used the word *parodontoclasia* as a generic term, because it was adopted by the Chairman of the Committee on Nomenclature of the American Dental Association. Dr. Anthony's arguments for the use of this word appeal to me. However, I have no intention to make an issue of terminology, while there is so much more important work to be done.

The cases of Dr. Hirschfeld are very interesting. Conditions such as he has shown occur also in people without endocrine disturbance. However, this disease category represents a most promising field which, from the dental standpoint, is practically unexplored.

We take cognizance of traumatic occlusion, but do not overemphasize its importance. The effects of occlusal strain upon the integrity of the investing tissues are pretty well understood. Research is concerned with new knowledge, not with the resurrection or popularization of old fallacies containing a modicum of truth. Homologues of the far-reaching claims and evangelical attitude of some of the protagonists of this hypothesis are to be found in the European literature of a quarter of a century ago. The status of traumatic occlusion as an etiologic factor in pyorrhea has been

thoroughly debated abroad. The results of such critical study will reach this country in time, and will probably establish the subject on a rational basis.

Dr. Merritt's question as to treatment I will answer somewhat at length:

Treatment. (*a*) *Preliminary observations.* The first step in the treatment of pyorrhea is tentative diagnosis. Thus we may judge that a mouth is essentially carious or pyorrhetic; that the pyorrhea is of the inflammatory or non-inflammatory species; that the condition is exogenous or endogenous. We may classify it clinically as traumatic, sialocalcic, sulcicalcic, granulative, crevicular, or parodontic. This superficial diagnosis is merely tentative. We may change our views of the case when the detailed examination is complete. We next proceed to measure and record the amount of destructive change which has occurred. Two general methods are available for this purpose—instrumental and roentgenographic.

In our earlier work these measurements seemed to afford sufficient data, and we made only general notations as to the hygiene and symptomatology of the gingivae and alveolar mucosae. To our surprise, however, we found that, in spite of the most refined methods of measurement, cases of pyorrhea (even advanced cases) might show no detectable change in a year's time. We then realized that the minute characters of the gingivae were delicate indicators of conditions, and have since included, in our examinations, careful examination and record of gingival stigmata. The classification of these appearances is at present in a developmental stage and the nomenclature tentative. The following observations and designations have proven useful:

(1) The Hinshaw stigma[30]—Arterial
Venous

(2) Gingival margin slightly blunted

(3) Facial or atrial gingival crescents—Bright red
Pink
Lavender
Purple

(4) Facial or atrial bands[31]—Narrow—Pink
Broad—Lavender
Purple

[30] This condition has been described by Dr. H. Corwin Hinshaw in "The Protozoa of the Human Mouth" (thesis written in partial satisfaction of the requirements for the degree of Doctor of Philosophy), awaiting publication. The form of the gingivus is normal, the marginal edge thin and almost colorless. About 1 mm. from this edge a narrow crescent may be seen—red in the case of arterial hyperemia, lavender in venous hyperemia.

[31] Crescentric marginal strips, one, two or occasionally more millimeters in width.

(5) Facial or atrial gingivae
and alveolar mucosae—Pink rash—Diffuse
Red rash—Irregular

(6) Gingivae—Smooth, firm
Sub-edematous
Edematous
Reddens under pressure from point of instrument
Retains imprint of point of instrument[32]
Bloated
Parchment-like[33]

(7) Gingivae—Wrinkled[34]
Lined
Incised
Fissured
Serrated
Lobulated

(8) Gingival marginal edge—Limp

(9) Gingival margin—Limp

(10) Gingivae—Festooned

Festoons { Narrow / Medium / Wide

Festoons { Upthrust / Everted / Flaccid

(11) Gingival papillae—Stippled
Smooth
Sub-red
Red
Lavender
Purple
Tip limp

[32] In the aged it may be noted that pressure (on the back of the hand, for example) produces an imprint which is only slowly obliterated, whereas the young tissues rebound at once.

[33] The disappearance of fluids of edema may leave a previously stretched "skin" unsupported, in which case it acquires the surface appearance of parchment, something after the manner of a deflated toy balloon.

[34] These are fine radiating wrinkles. They can sometimes be detected only after careful scrutiny or by playing reflected light across them.

Parchment-like
Bloated
Saddled[35]
Cloven
(12) Injected arteriole—Size
Position

Record should be made of the hygiene of the gingivae before studying the stigmata, as this necessitates cleaning and drying of the tissues. The amount of *materies alba* is employed as indicative of the hygiene. The following notations are sufficient: Excellent, Good, Medium, Fair, Poor, Very Poor.

The observations and designations given above have proven indispensable in our research work. It is obvious that examinations of this type must be made by the same person. The significance of the terms used and the minute characters to which they refer can be definite and remain so in the mind of a given observer. The important point is that the designation shall recall to the mind in the future the exact condition which it was intended to record.

Other terms may be found more descriptive, clear, or useful to other observers or those suggested here may in some cases portray different pictures to those in the mind of the writer, yet since they are purely comparative they appear amply to serve the purpose.

(*b*) *Instrumental examination.* This method is concerned with the measurement of detachments of the soft tissues from the cementum of the root of the tooth. The procedure has been presented in detail in published articles.[36,37] It should be stated, however, that since according to Gottlieb's findings the bottom of the sulcus previous to and during the third decade of life may be crown-wise of the gingival line, there may be some objection to the use of this line as a landmark. However, in those cases of pyorrhea—rare in our experience—where the dehiscence has not reached the gingival line,[38] we can estimate the distance of the bottom of the sulcus therefrom and indicate as a negative detachment, thus: $-\frac{1}{2}-1$.

[35] Marked by loss of tissue at the center, facio-atrially. Precedes the cloven condition where only facial and atrial horns remain, which may be drawn out from the embrasure.

[36] Simonton, F. V. Examination of the mouth—with special reference to pyorrhea. *Journal of the American Dental Association*, 1925, v. 12, pp. 287–295.

[37] Simonton, F. V. Technic involved in measuring destructive changes of investing tissues of the teeth. *Ibid.*, pp. 448–453.

[38] This sometimes is the case on the distal or mesial of cuspids, even in cases where the disease has progressed considerably in other positions.

(*c*) *Roentgenographic examination.* We found it necessary to design an instrument which would make it possible to duplicate roentgenographs with mathematical precision at any desired interval of time. Such an instrument was perfected several years ago, has been in use ever since, and has proven practical and adequate. Its construction and use is described in a recent article. (See footnote 20, on page 251). This method enables us accurately to determine changes which may take place in the level of the alveolar crests or in cancellation of bone. Measurements may be compared by direct calipering of the films. Full mouth roentgenographs are necessary.

We have also devoted some effort to mouth photography, and have obtained results both in black and white and in the natural colors which will be reported in the near future.

In a number of cases, in a whole year's time we have found no evidence of change which could be detected by these methods. This is a remarkable demonstration of the potential chronicity of pyorrhea, and strong evidence that spontaneous latency or abortion may occur. The level of the crest of a previously much retracted alveolus might not vary a fraction of a millimeter. It may require six months time for the obliteration of the fine wrinkling of a gingival margin, as it passes into a sub-edema—or the wrinkling may remain unchanged through an entire year. On the other hand, the degree of involvement possible in young cases indicates that retrogressive changes may occur rapidly, although I have not yet had opportunity to follow such cases through a suitable period and tabulate the evidence. Redundant tissue, lobulated masses or granulated tissue, may develop in a few days, weeks, or months.

(*d*) *Abbreviated form of examination.* The form of examination described above has been developed in response to our research needs, but is entirely too time-consuming for practical purposes. In certain cases it may take as long as sixteen hours to measure detachments and record stigmata alone. For routine use, we suggest partial examination at the following sites:

Upper central incisor	Facial aspect
Upper lateral incisor	Mesial aspect
Upper cuspid	Atrial aspect Distal aspect
Upper first bicuspid	Mesial aspect

Upper first molar	Facial aspect
	Atrial aspect
	Distal aspect
Lower central incisor	Facial aspect
	Atrial aspect
	Mesial aspect
	Distal aspect
Lower first bicuspid	Atrial aspect
	Mesial aspect
	Distal aspect
Lower second bicuspid	Facial aspect

Also a facial and atrial aspect of a third molar.

It is unimportant whether the right or left teeth be selected, but whichever are chosen should be adhered to, except where one is missing, when the corresponding one of the opposite side may be substituted. These positions are selected in accordance with the sites of greatest or least detachment, as shown by Fleming.[39] The third molar is included because of its late eruption. These selections also conform well with areas of varying bone mass, of varying liability to trauma, and of varying liability to stagnation of debris.

On the basis of our several examinations we are now in a position to estimate the involvement and to judge the degree of intensity. We also have a record from which we can measure the rate of progress of the disease or the effectiveness of our treatments.

(*e*) *Retrogressive changes.* There are a considerable number of factors which may cause retrogressive changes in the investing tissues. A variety of such factors may also be operative in the individual case. This complex etiology of pyorrhea requires careful search for these factors, and often in the same mouth different teeth become different patients. The complexity of the problem or the classification will not lead to confusion, however, providing the logic of the scheme is understood.

(*f*) *General physical condition.* During the course of the detailed dental examination we should seek through observation and conversation to learn what we can of the patient's general physical condition. We may find it necessary to refer the patient to a physician. The general data which are

[39] Fleming, Willard C. Localization of pyorrheal involvements. *Dental Cosmos*, 1926, v. 68, pp. 538–541.

of most interest to us are such as have to do with recent change of weight, general disease or debility, condition of other epithelial appendages, indications of feebleness of growth, stigmata of rickets (as pertaining to possible hypoplasia of the alveolar process), and indications of circulatory disturbances. Information relating to the following has proven useful:

Recent change of weight
Hemoglobin
Blood pressure
Wassermann
Complexion
Surface capillaries
Skin, condition of
Hair—quantity
quality
Eyes—appearance
conjunctiva
sclera
cornea
iris
retinal vessels
Finger nails
Finger nails, under
Tonsils
Pharynx
Diseases
Accidents
Operations
Bowels
Drugs
Alcohol
Tobacco
Chest—shape
development
Glands—anterior cervical
posterior cervical
Thyroid
Reflexes
Stigmata of rachitis
Dwarfing
Head
Chest
Spine
Long bones—epiphyses
diaphyses
Feet
Feebleness of growth
General physical condition.

(*g*) *Mouth hygiene.* The next step consists in cleaning and scaling the teeth, and instructing and training the patient in mouth hygiene. Most debris is capable of irritating the gums, and maintains a slow fire, as it were. The burning away from this cause should be stamped out as quickly as possible. The two most effective ways to accomplish this are by the tooth brush and massage. A practical and effective method of brushing the teeth and gums has been described and carefully illustrated in a recent article.[40] It is not at all uncommon to see mouths in which the investing tissues of the more accessible teeth, which have been kept clean, are sound, while those in the back of the mouth, where the effort at hygiene was unsuccessful, are ravaged. These cases are particularly tragic, because preventable. Cleaning about a front tooth will not save a dirty back tooth. The periodontist must teach the patient how to effectively clean *all* of his teeth and must insist that he do it.

(*h*) *Relief of excessive occlusion.* Obvious points of traumatic occlusion should be relieved when observed. Scientific adjustment of the occlusion, however, requires the mounting of casts on an anatomical articulator, and a survey of the case from the standpoint of the expert crown-and-bridge

[40] Simonton, F. V. How to clean the teeth and gums. *Better Health* (San Francisco), October, 1927.

prosthetist. Any general grinding of the cusps without such preliminary study will be ineffectual and may be harmful.

(*i*) *Deep scaling.* A method of scaling the roots of the teeth is described in a previous article.[41] It should be repeated that the removal of deposits is not the only function of this technic—the stimulation afforded by the massage effect of the instrument is of equal importance.

(*j*) *Massage of the gums* by the dentist is a valuable treatment. The patient should also be taught how to do this for himself. We are now working on a vibratory device to be used in the engine by the dentist. Preliminary trials have been gratifying.

(*k*) *Habits*, such as grinding the teeth, clenching the teeth on a pipe stem, etc., should be controlled.

(*l*) *X-ray, etc.* We have recent evidence that x-ray therapy is valuable for local stimulation of the tissues. The usefulness of the ultra-violet ray has not yet been determined. We have gotten good results with it in the treatment of skin lesions, but nothing definite in pyorrhea. It may prove more effectual if the gums are first painted with eosin. We are testing this out. Cod liver oil may be prescribed both for vitamin content and for specific dynamic effect. Dr. Prinz has recommended Fowler's solution for the purpose of producing recalcification of halisteretic bone. Dr. Gottlieb is also working with arsenicals.

(*m*) *Diet.* The diet should now be investigated. The best method I know for this purpose is that employed by Dr. M. R. Jones, requiring the patient to keep a list of all articles eaten during a week's time, including the quantity of each. Such a list will be more accurate than any information which can be obtained off-hand from the patient. Study of this list will permit the estimation of the caliber of the diet from the standpoint of vitamins, mineral salts, and acid-base balance. The recommendations of McCollum[42] for a satisfactory diet are simple and easily remembered. He advises:

(1) Approximately a quart of milk a day.
(2) Once a day a liberal serving of greens or potherbs.
(3) A salad twice a day.

The acid-base balance of the diet should now be calculated. If the patient has been running strongly to one side or the other, we are in a posi-

[41] Simonton, F. V. A conservative method for the treatment of pyorrhea. *Journal of the American Dental Association*, 1925, v. 12, pp. 197–202.

[42] McCollum, E. V., and Simmonds, Nina. Food, Nutrition and Health. Published by the authors. Baltimore, Md. (Johns Hopkins University).

tion to manipulate these factors, being guided by clinical results. Care must be taken, however, not to plan an acid-base balance which would be in conflict with the systemic needs of the patient. Acid producing foods include meats, dairy products, breads, and cereals. Alkali producing foods include beans, peas, potatoes, citrus fruits, and green vegetables.

(*n*) *Vaccines.* Autogenous vaccines seem to be the most rational type for use in inflammatory pyorrhea. We have followed the method of Dr. Beckwith, aspirating fluid from the tissues. This material is inoculated into a culture tube, a vaccine prepared, and used in the customary dosages.

(*o*) *Surgical methods.* In advanced cases, or where immediate elimination of infection is required, it may be necessary to resort to surgical procedure. Windows may be cut at the sites of the depths of pockets and the necrotic tissue curetted away. The open flap operation may be employed, or occasionally excision of pockets may be justified.

THE ERUPTION AND GROWTH OF THE PERMANENT TEETH

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I. THE AGE OF ERUPTION

In connection with an extensive study of the growth of children, conducted at Harvard University under the direction of Professor Walter F. Dearborn, certain data have been obtained which, it is believed, are of interest to those engaged in dental practice. The unusual opportunities afforded for obtaining uniform dental records from a large number of unselected school children make it appear desirable to add another table to those already found in the literature giving the age of children at the time of the eruption of the various teeth.

The table that follows is based on the records obtained from 7,689 dental examinations, 3,863 of which were made on boys and 3,826 on girls. The examinations were made by J. Murray Gavel, D.M.D., of Boston, assisted in about 5 per cent of the examinations by William Jones, D.M.D., of Newton Center, Mass. Thus uniformity and exactness of procedure was assured. The writer is greatly indebted to Dr. Gavel for reading the manuscript and for constructive suggestions.

The race factor was eliminated by including only children who were of North European (excluding Jewish) descent, as determined by the birth place of the parents. The sexes were treated separately. Ages were recorded to the nearest month, the age groups used covering a period of six months. The mid-point of an age interval is used to designate the group. Thus the age group six years and three months

(6–3) includes all those whose ages were recorded as six years and no months (6–0) to six years and five months (6–5) inclusive; etc.

The upper section of *Table 1* gives the age at which 25 per cent, 50 per cent, and 75 per cent of the various teeth have erupted in boys. The lower section of the table gives the same for girls. The 25th percentile or lower quartile point (Q_1) is the age at which 25 per cent of the children have the tooth in question present, while in the other

TABLE 1

The age of children at the time of eruption of the permanent teeth

TEETH	LOWER JAW			UPPER JAW		
	Q_1	M	Q_3	Q_1	M	Q_3
Boys						
1, or third molars	—	—	—	—	—	—
2, or second molars	11– 6	11– 8	12– 9	11– 6	12– 2	13– 2
3, or first molars	5– 9	6– 2	6–11	5–11	6– 4	7– 1
4, or second premolars	10– 3	11– 4	12– 6	9–11	11– 1	11–10
5, or first premolars	9– 7	10– 7	11– 7	9– 1	10– 4	11– 5
6, or canines	9–11	10– 8	11– 5	10– 9	11– 6	12– 6
7, or lateral incisors	7– 0	7– 7	8– 2	8– 0	8– 5	9– 2
8, or central incisors	5–11	6– 3	6–11	6–10	7– 4	7–11
Girls						
1, or third molars	—	—	—	—	—	—
2, or second molars	10– 7	11– 5	12– 2	11– 3	12– 1	12–11
3, or first molars	5– 9	6– 0	6– 5	5–10	6– 2	6– 8
4, or second premolars	9–10	11– 1	11–11	9– 9	10–11	11–10
5, or first premolars	9– 3	10– 1	10–11	9– 0	9–11	10–11
6, or canines	9– 1	9– 8	10– 5	10– 1	11– 1	11– 8
7, or lateral incisors	6– 8	7– 3	7– 8	7– 6	8– 0	8– 8
8, or central incisors	5– 8	6– 1	6– 5	6– 7	7– 1	7– 7

75 per cent of the cases the tooth has not yet broken through the gums. The median (M) gives the age at which the tooth has erupted in just one half or 50 per cent of the cases. The 75th percentile or upper quartile point (Q_3) indicates the age at which the tooth has erupted in 75 per cent of the cases. Thus the first column in *Table 1* (headed Q_1) gives the age at which one fourth of the children have erupted each tooth on the lower jaw; the second column (headed M), the age at which half the children have erupted each tooth; etc. A comparison

of the upper and lower sections of *Table 1* shows the girls to be appreciably in advance of the boys, age for age.

Figures 1 and *2* are percentile curves which give the percentage of boys and girls, respectively, of each age having each tooth erupted. The vertical scale on the left represents the age; the horizontal scale at the bottom the percentiles, and each curve one tooth. Should one wish to know what proportion of boys have each tooth erupted at the age of six years and nine months (6–9), *fig. 1* would be read as follows: First locate the horizontal line labeled 6–9 and follow along it until it intersects the first curve (7), which is for the lower lateral incisor, and then read the horizontal scale directly below, which will be found to be approximately 17, showing that 17 per cent of the boys of this age have this tooth erupted. Following the line out to the next curve (8), it will be found that 23 per cent have the upper central incisor erupted. Continuing this process, it is learned that 60 per cent have the upper first molars, 68 per cent the lower central incisors, and 69 per cent the lower first molars. The fact that the curves for all the other teeth are above the 6–9 line indicates that none of the boys at this age had these teeth erupted.

It is a well known fact that there is a marked tendency for the teeth to erupt in pairs. It must, however, be emphasized that this is only a tendency and by no means an invariable fact. Frequently several months elapse between the eruption of one tooth and its mate on the other side of the jaw, and during this time three or four other teeth may erupt.

It does not appear to have been definitely determined whether there is a constant difference in the time of eruption of the teeth on the left side and on the right side of the jaw. As early as 1859 Tomes[1] stated that the teeth erupt earlier on the left side than on the right side of the jaw. More recently Bean[2] concluded from his data that some teeth erupt slightly earlier on the left side, while others erupt slightly earlier on the right. In the hope of settling this question, percentile charts have been constructed showing the difference between the right and the left side of the jaw. Samples of these curves, which are typical of the others, are shown in *fig. 3*. With the exception of the upper first pre-

[1] Tomes: A system of dental surgery: Lindsay and Blakiston.

[2] Bean: Stature and eruption of the permanent teeth of American, German-American, and Filipino children. *American Journal of Anatomy*, 1914–15, xvii, p. 113.

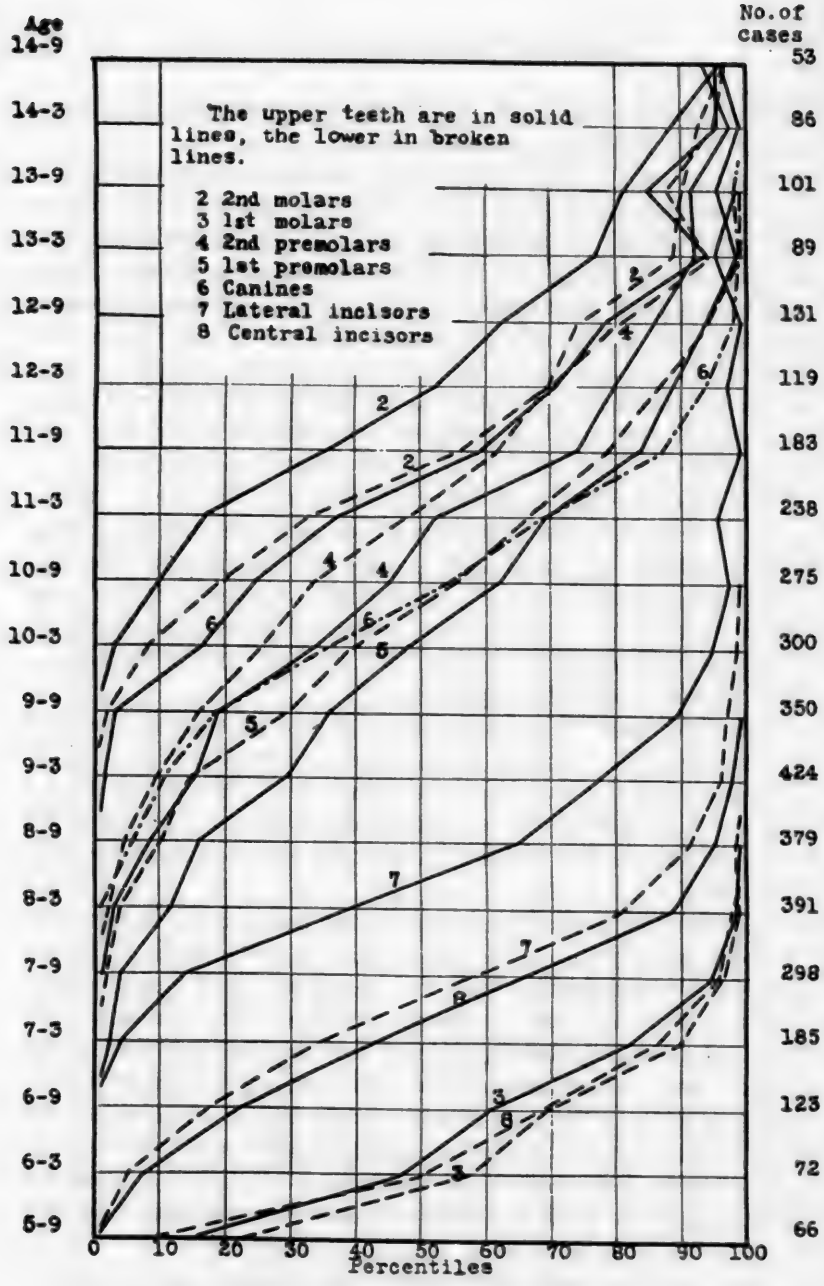

Fig. 1. Percentage of teeth erupted at various ages—boys

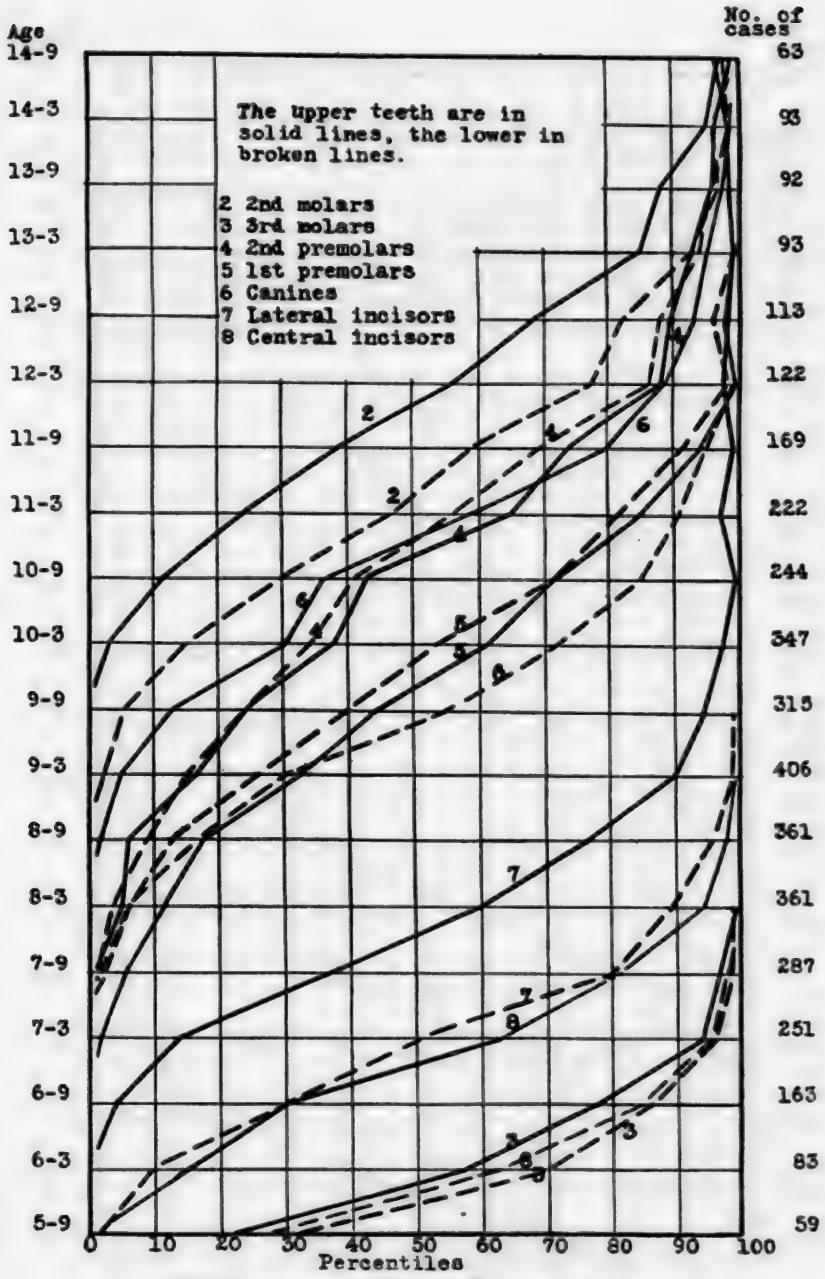

Fig. 2. Percentage of teeth erupted at various ages—girls

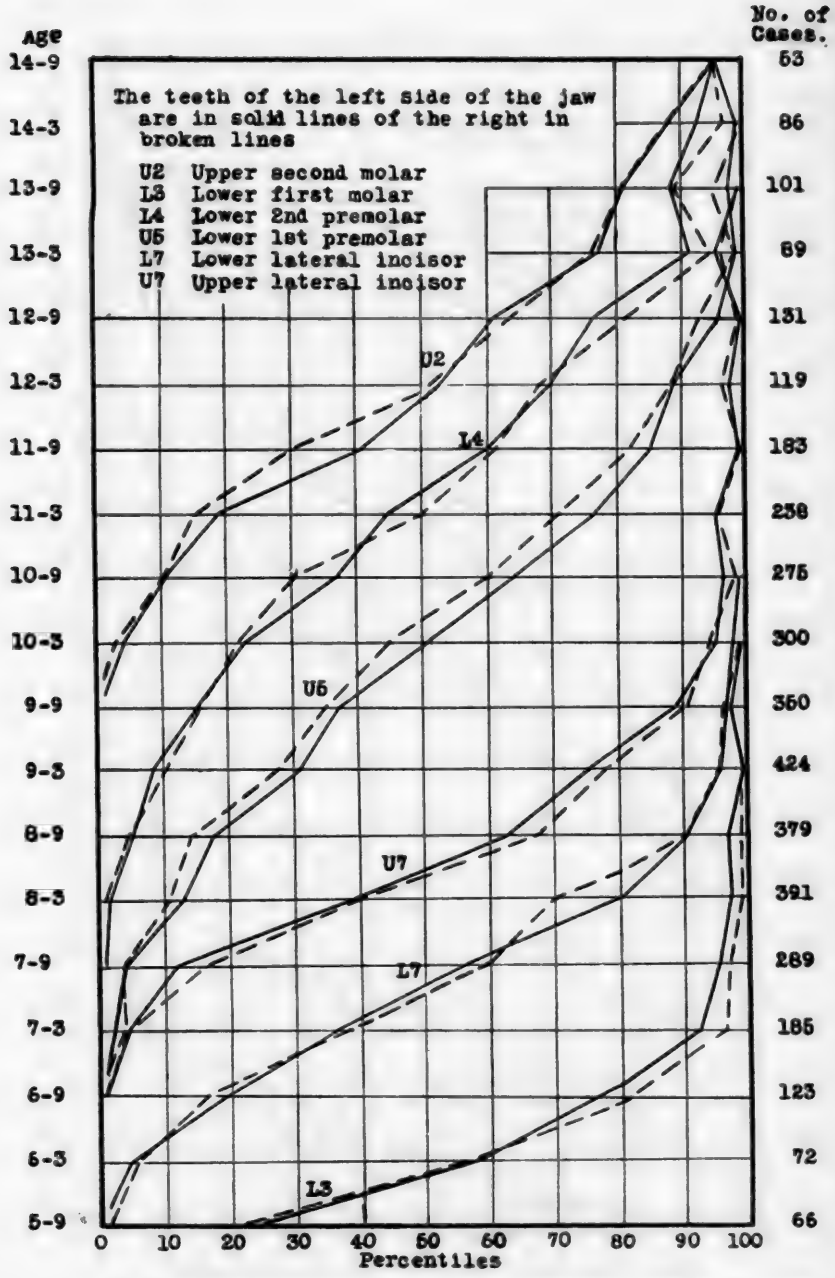

Fig. 3. A comparison of the age of eruption of the individual teeth of the right side and the left side of the jaw—boys.

molar, no constant tendency is seen for a tooth on one side of the jaw to erupt earlier or later than its mate on the other side. According to the data under consideration, the right upper first premolar erupts about two months in advance of the left. Since there is no indication of a constant difference in any of the other pairs of teeth, it is probable that the difference found in the premolar is due to a chance variation in the data used.

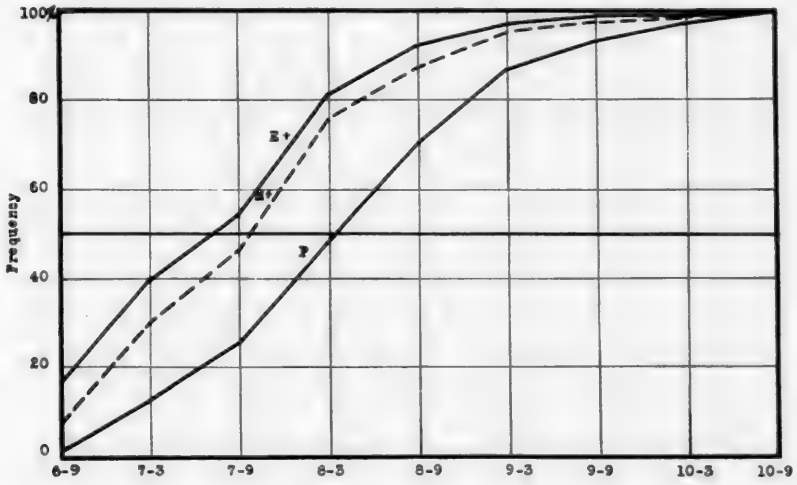

Fig. 4. Percentage of boys who have the lower lateral incisor erupting or further advanced (E+), half way through (H+), and in full occlusion (P).

II. RATE OF GROWTH

In connection with the study of the growth of children, it became desirable to learn the rate of growth of the teeth after they break through the gums. Neither a careful search through the literature nor consultation with those engaged in dental practice yielded satisfactory information. The most frequent estimate made by the dentists was about four months. They admitted, however, that this was scarcely more than a guess.

In the dental examinations of this study each tooth was recorded in three stages of growth: "erupting" (E), "half way through" (H), and

"present" (P). From these records the rate of growth of a tooth was estimated by calculating the age at which 50 per cent of the children had the tooth in question just erupting or further advanced, half size or larger, and in occlusion. The difference between the age at which 50 per cent of the children had reached the stage "erupting," and the age at which 50 per cent had reached the stage "present," gives a rough indication of the time taken to proceed from the first stage to the last. The results for several of the teeth are given in *Table 2*.

Figure 4 is presented as a sample showing in graphic form the percentage of cases in each age-group that have the lower lateral incisor at the various stages of growth. These figures are for boys only. It is not claimed that they are exact. Owing to the fact that a tooth may remain in the stage "erupting" for several months, it is probable that

TABLE 2

The rate of growth of teeth, in months

TOOTH	TIME FROM E TO H	TIME FROM H TO P	TIME FROM E TO P
Upper first incisor	2.75	5.82	8.57
Lower first incisor	2.58	5.66	8.24
Upper second incisor	2.94	6.11	9.05
Upper first premolar	2.27	1.84	4.11

the rates of growth are underestimated. However, there is apparently no more reliable information available. The estimates are considerably above those given by dentists and are probably more nearly correct. The incisors appear to grow more rapidly than the premolars. The calculations were not made for the other teeth, for the importance of taking repeated measurements at short intervals of time on the same children had become evident, and Professor Dearborn initiated a series of monthly measurements on about 500 children. These measurements were begun in March, 1925, and a few of the teeth were still in the process of growing in November, 1926, when the last examinations were made. The results of this study are not yet ready for presentation, but they indicate that the estimates given above are too short. A striking result is the great irregularity in the rate of growth. Some teeth completed their growth in a couple of months,

while others had not reached maturity after eighteen months. There appears to be a tendency for some types of teeth to grow more rapidly than others, but there is also great variation in the same type of teeth in different individuals.[3]

[3] This paper was presented at the sixth annual meeting of the International Association for Dental Research, Washington, D.C., March 28, 1928. A more extensive study of the dental data, with a detailed description of their collection and treatment, is to be published by the Harvard University Press, in the near future, under the title: "Dentition as a measure of maturity."

NEW EVIDENCE OF MAN'S RELATIONSHIP TO THE ANTHROPOID APES

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The subject of man's blood-relationship to the anthropoid apes is once more to the fore, and with a new significance. Scarcely had the echoes of the Dayton trial died away when the question was brought forward anew with denial of such relationship by one of the foremost scientific men in America.

Professor Henry Fairfield Osborn, President of the American Museum of Natural History, is known throughout the world for the untiring energy and the great ability he has shown in tracing the origins and relationships of many forms of animal life. The results of his researches have been published in many scholarly volumes. In his books and articles he has not only covered the whole ground of the origin of life and the history of the doctrine of evolution, but he has also entered the very controversial field of the relations of science and religion. Because of the very broad scope of his work and the high standing which this work has given him, any pronouncement which he may make, upon any subject within a purview so wide that it almost covers the entire field of the science and philosophy of evolution, demands and receives the most serious attention. When, therefore, he projects into a mental atmosphere already highly charged with emotional sentiment on the subject the opinion that there is no blood-kinship between man and ape, in direct opposition to views which have become firmly established in the minds of a great majority of the foremost scientific men in the world, the whole great problem of man's origin from lower forms of life should at once be lifted above the controversies of Main Street, the cheap bickerings and scoffings of Fundamentalists and blatant, aggressive atheists, into the more serene atmosphere of purely scientific discussion.

The unfortunate thing, however, about the whole situation is that

the great army of those who are working with the energy and determination characteristic of fanatics to control education and confine it

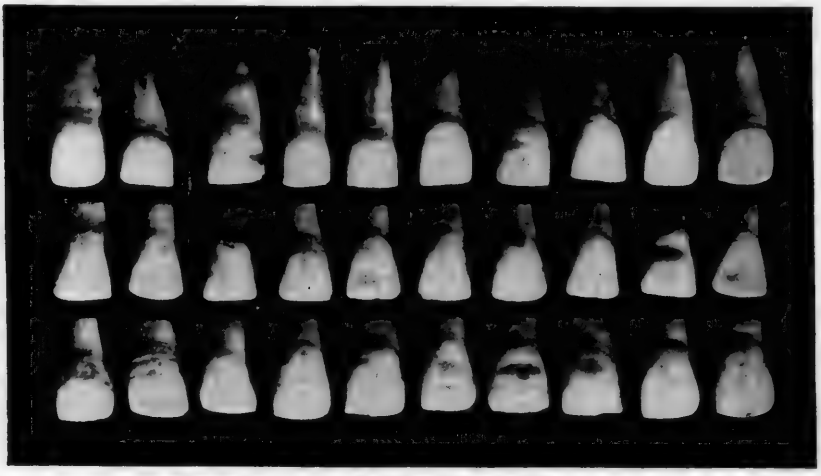

Fig. 1. Three distinct forms of human central incisors; Classes I, II, and III

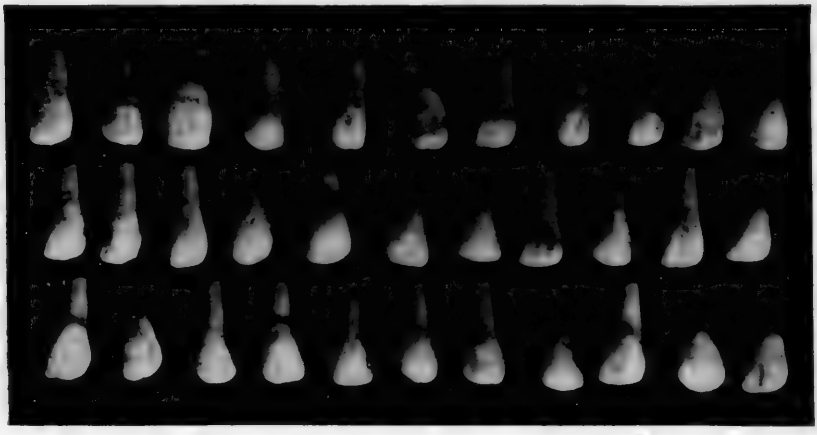

Fig. 2. Variations that occur in the upper lateral incisors

within the bounds of narrow, sectarian interests will be much enheartened and stimulated to even greater efforts by Professor Osborn's

recent pronouncements. There was already grave cause for anxiety in the present situation. Few people seem to be fully aware of the implications of the Fundamentalist reactionary attitude of mind with relation to the whole organization of human society and the effects of this attitude of mind in hindering human progress.

Believing, as I do, that it will soon be evident that certain statements in Professor Osborn's recent book, "Man Rises to Parnassus," will be used with great effect by the Fundamentalists in their efforts to control education in the common schools, I propose to challenge the opinions which he has expressed concerning man's relationship to

Fig. 3. Sandwich Islanders; Classes I, II, and III. See *figs. 4, 5,* and *6*

the ape with evidence of a new and convincing character. And as this evidence is confined entirely to researches into homologies of tooth form, it very properly appears in the *Journal of Dental Research.*

Quite a number of years ago, while living in London, deeply impressed with the lack of critical information concerning tooth form in relation to race, and mindful of what Huxley had said about the importance of the teeth in researches of this character, I determined to take up this study, with laudable curiosity to see what might come of it. I had long observed that the most obvious variations in human teeth occur in the upper central incisors, and the first step in my investigation was to secure a large collection of these teeth from

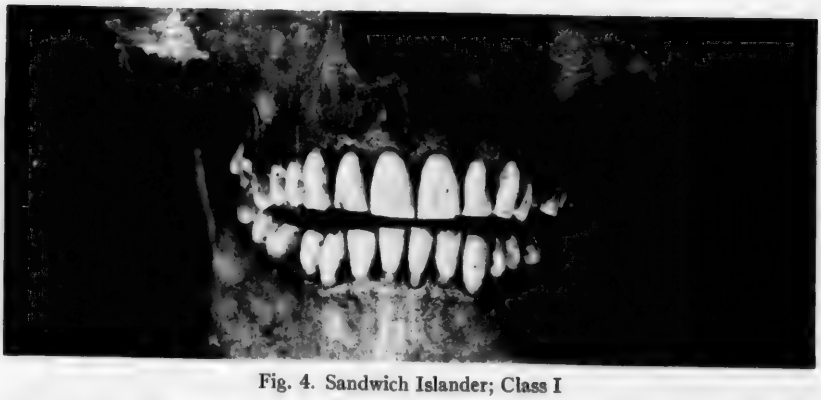

Fig. 4. Sandwich Islander; Class I

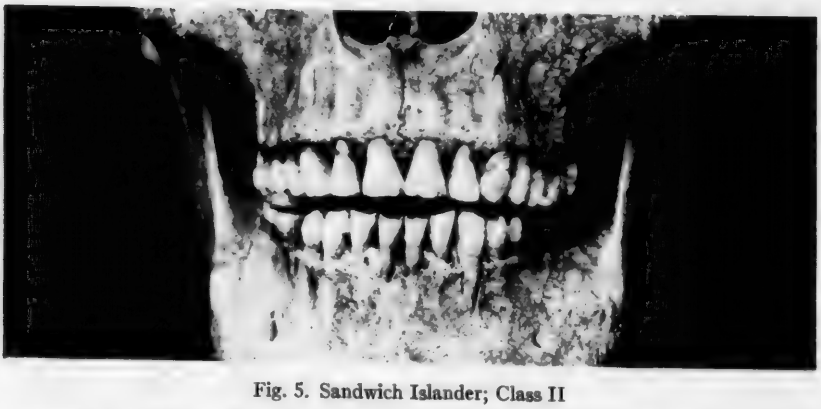

Fig. 5. Sandwich Islander; Class II

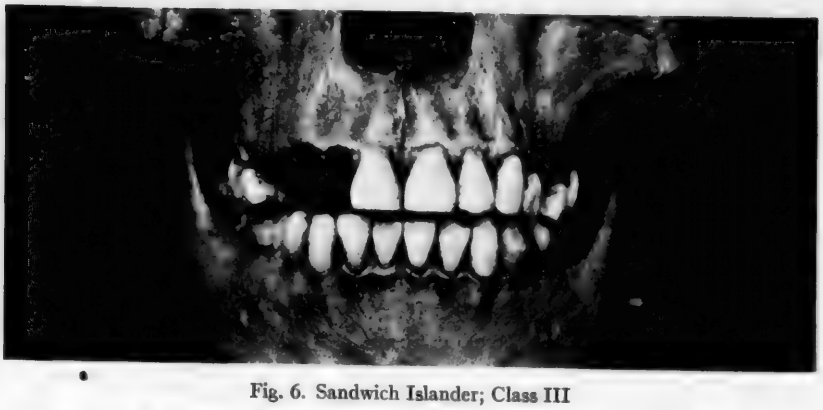

Fig. 6. Sandwich Islander; Class III

Figs. 4-6. Sandwich Islanders; Classes I, II, and III. Enlargements of *fig. 3*

Fig. 7. Modern Chinese; Class III

Fig. 8. Modern Hindoo; Class III

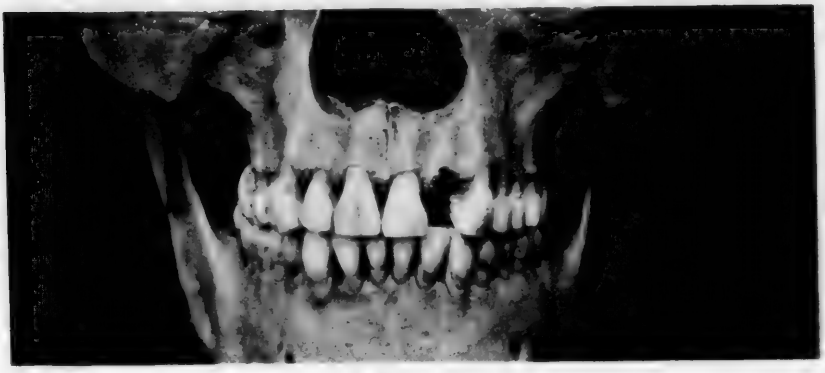

Fig. 9. Kaffir; Class III

Figs. 7–9. Typical forms of Class III

different parts of the world. In the course of a year I found myself in possession of more than a thousand specimens. A superficial examination showed wide and marked variations in form *in all races*, with only vague indications of racial peculiarities.

I then set about sorting and arranging these teeth according to the most striking peculiarities of form, placing together those showing the greatest resemblance.

The method of arranging these teeth for convenient examination was to press the lingual surfaces into sheets of black wax. The outline of these teeth was thus very clearly seen against a black back-

Fig. 10. Tasmanians; Classes I, II, and III. See *figs. 11, 12,* and *13*

ground. It soon became apparent that there are *three* very distinct forms of human central incisors. These three forms are plainly shown in *fig. 1*. In the upper row we see that the proximal sides of the teeth are parallel for one-half or more of the length of the crown. The most striking effect of this in the mouth is to reduce the interproximal space, as the teeth are in contact for more than one-half the length of the crown. In the second row we see the proximal sides of the teeth rapidly converging from the incisal edge to the cervical or root end of the crown. In the third row the first distinguishing peculiarity that attracts our attention is a double curve most frequently appearing on the distal proximal surface of the crown, but sometimes seen on the

Fig. 11. Tasmanian; Class I

Fig. 12. Tasmanian; Class II

Fig. 13. Tasmanian; Class III. Here, in this primitive and comparatively unmixed race, we find abundant proof that there is no racial type of tooth. Compare *fig. 11* directly with *fig. 13.*

Figs. 11–13. Tasmanians; Classes I, II, and III. Enlargements of *fig. 10*

mesial surface and occasionally, though not often, on both mesial and distal surfaces.

Distinguishing these teeth as of the first, second and third class, respectively, there are also other minor but distinct features peculiar to each of these forms to which attention should be directed. The labial surfaces of the crowns in Class I are usually very regularly curved both mesio-distally and cervico-incisally. The labial surfaces of those in Class II are generally much flatter than in either of the other classes and are often shovel-shaped, the central portion of the labial surface often being lower than the sides. I cannot recall that

Fig. 14. Native Australians; Classes I, II, and III. See *figs. 15, 16,* and *17*

I have ever found this peculiarity in any except teeth of this class. Curves of marked gracefulness, including the double curve mentioned, distinguish the teeth of Class III.

Naturally, as seen in the mouth, the interproximal spaces are very marked in Classes II and III when compared with Class I. It should be stated here that the teeth shown in *fig. 1*, represent a minority of all human central incisors. They are the most severe of the typal forms. The great majority, as we should expect, are various blendings of these typal forms. But in most teeth one or other of the typal forms is clearly dominant. All of the teeth in any given set partake more or less of the typal characteristics of the upper central incisors,

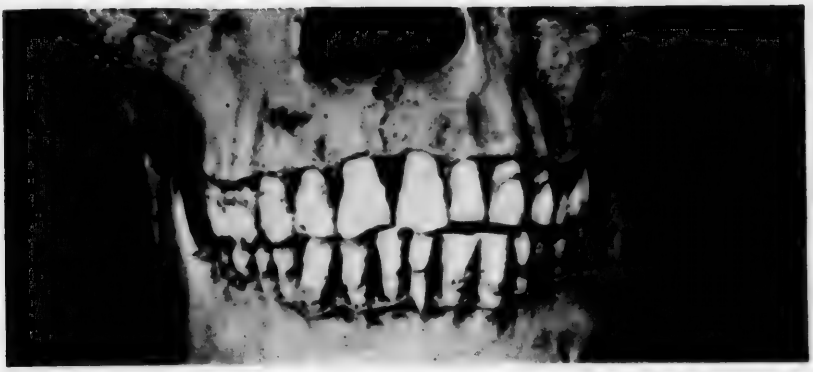

Fig. 15. Native Australian; Class I, slightly modified

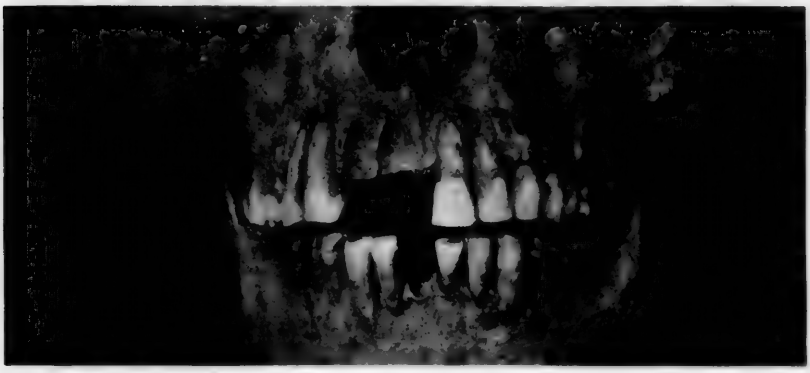

Fig. 16. Native Australian; Class II

Fig. 17. Native Australian; Class III

Figs. 15–17. Native Australians; Classes I, II, and III. Enlargements of *fig. 14*

but one who has not given long study to the less noticeable differences in teeth would hardly recognize these typal features at first.

The greatest variations in human teeth occur in the upper lateral incisors and in the third molars. How striking these variations are is well shown in *fig. 2*. Such marked variations are never seen, so far as I know, in the teeth of animals or in the more primitive races of mankind. There is much here suggestive of other phases of evolution that it is not within the province of this paper to treat.

When I had proceeded thus far in my investigation, my next step was an examination of the skulls in the large and fine collection of the

Fig. 18. Ancient Egyptians; Classes I, II, and III. See *figs. 19, 20,* and *21*

Museum of the Royal College of Surgeons of London. For some time, through the kindness of the head of this institution, Professor (now Sir) Arthur Keith, I had enjoyed the free run of this museum. Here I found in all racial or people groups represented, when the group contained any considerable number of skulls, the three types of teeth which I had isolated in my collection. The racial types of teeth which I had expected to find were non-existent. Instead, all races have three types of teeth, with certain minor racial variations. Subsequent study in other large museums and more than fifteen years' experience in the examination of teeth in the mouths of living people have established this discovery beyond all doubt.

Fig. 19. Ancient Egyptian; Class I. Perfect typal specimen

Fig. 20. Ancient Egyptian; Class II. Slightly modified and worn

Fig. 21. Ancient Egyptian; Class III. Perfect typal specimen

Figs. 19–21. Ancient Egyptians; Classes I, II, and III. Enlargements of *fig. 18*

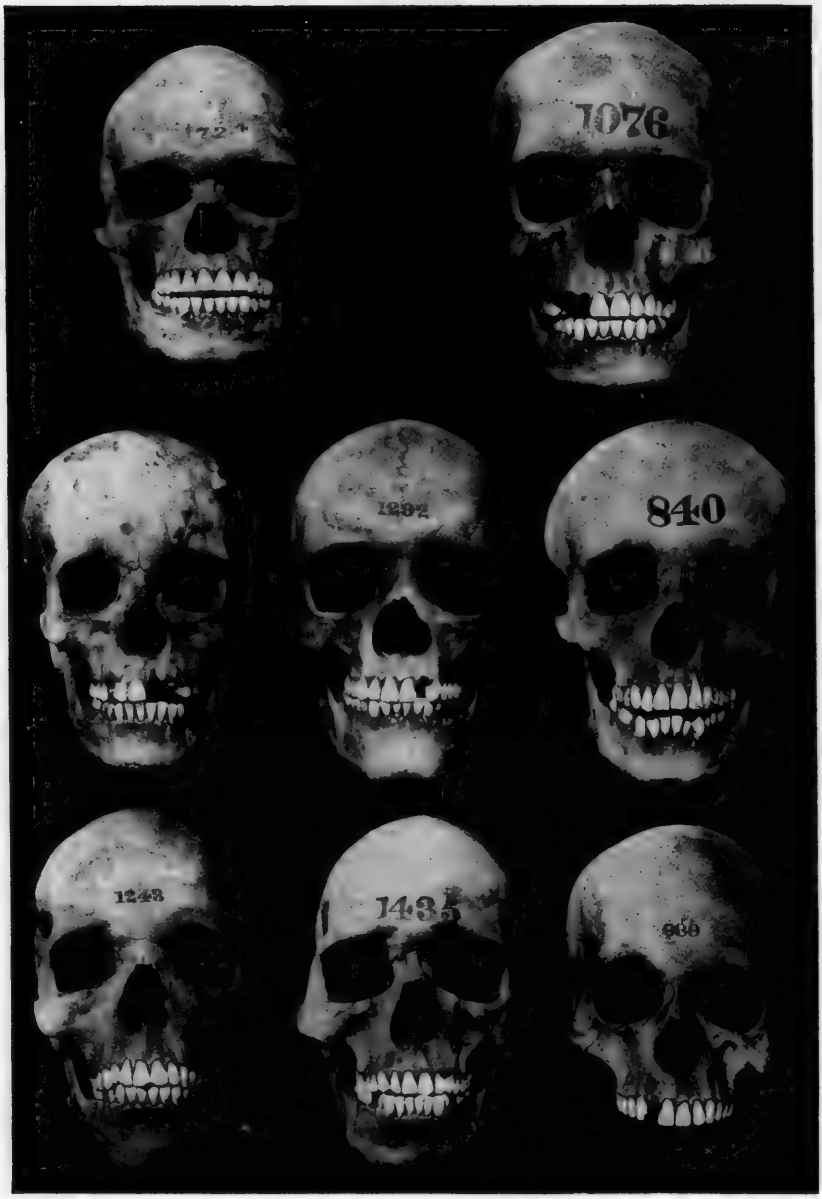

Fig. 22. All of Class III: Australian, Sandwich Islander, Ancient Egyptian, Kaffir, Chinese, African, New Hebridean, Hindoo.

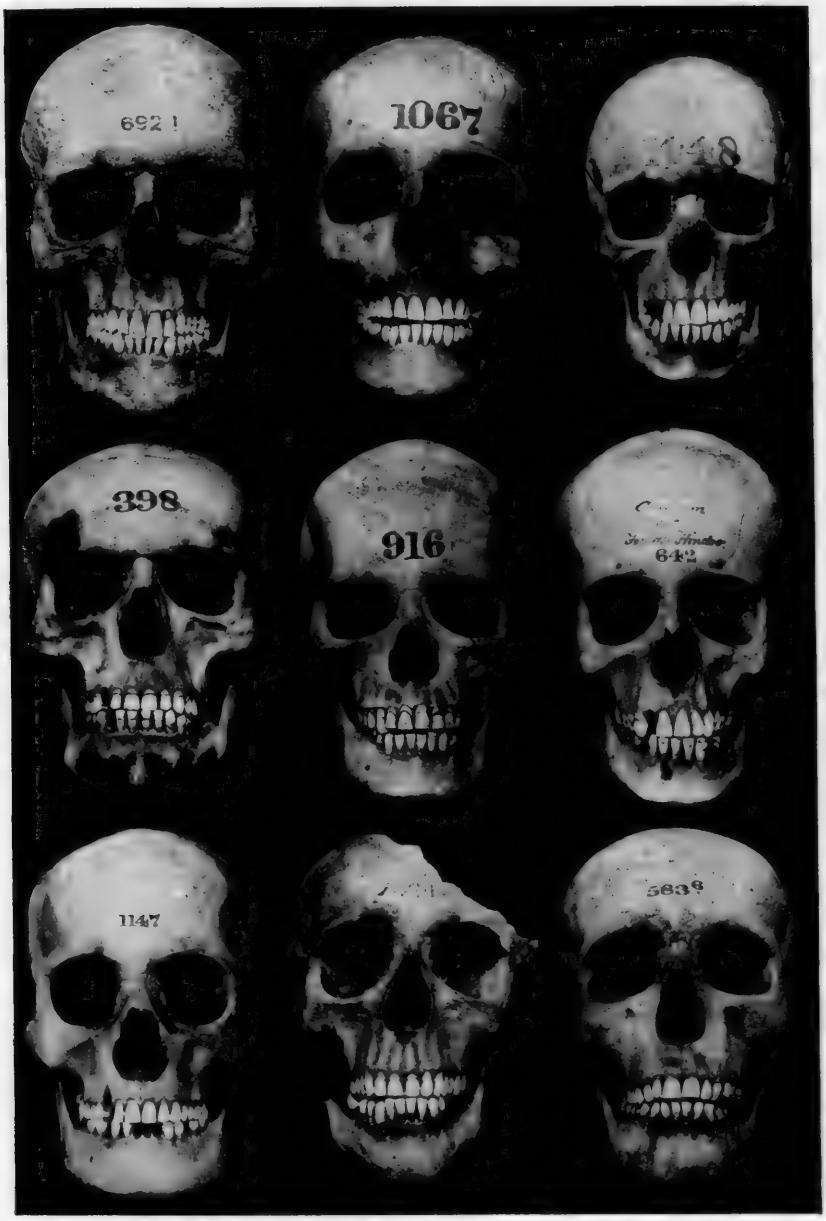

Fig. 23. All of Class I: Spaniard, Sandwich Islander, New Hebridean, German, Javanese, Hindoo, Fiji, Italian, Ancient Egyptian.

The further preeminently significant fact that these three types of teeth, even more strongly marked in their leading characteristics, are found in the gorilla, the orang-utan and the chimpanzee has such a direct bearing on the controversy now in progress as to the prehuman ancestors of man that it seems an opportune time to call the attention of the scientific world to the facts herewith presented.

Let us now examine a series of photographs for proofs in detail of the foregoing statements.

In a miscellaneous collection of central incisors (*fig. 1*) we have seen the three strongly marked types of teeth. We now proceed to show these same types in the skulls of different races or peoples.

We begin with three skulls of the Sandwich Islanders (*fig. 3*). The skull on the left, marked 1067, exhibits teeth of Class I, having proximal sides that are nearly parallel for half or more than half their length. In the middle skull, marked 1090, the teeth are of the tapering sort, Class II, characterized by sharply converging proximal lines and, consequently, by wide interdental spaces. The skull on the right, marked 1076, shows the double curved line on the distal proximal surface, Class III, also with wide interdental spaces. All of these characteristic features are much more clearly seen in the enlarged illustrations, *figs. 4*, *5*, and *6*, showing the jaws and the teeth only of the same specimens.

It should be understood that all the special features of the three types of teeth which I am describing are much more clearly seen in the skulls, where one has the opportunity of examining the teeth from different points of view, than in photographs.

However, the peculiar form of the Class III tooth, with its well-marked double curve on the distal surface, is a feature very well shown in *figs. 7*, *8*, and *9*, where the teeth are slightly separated, thus permitting the entire outline to be clearly seen. Upon studying these specimens closely it seems almost incredible that anthropologists and students of comparative anatomy should have made the mistake of supposing that there is any single form of tooth characteristic of race. Evidently, as I shall presently show, the mistake has been due to lack of close critical study and the examination and comparison of too few specimens.

Figure 10 shows three skulls of the recently extinct Tasmanians.

Fig. 24. New Hebridean; Class I

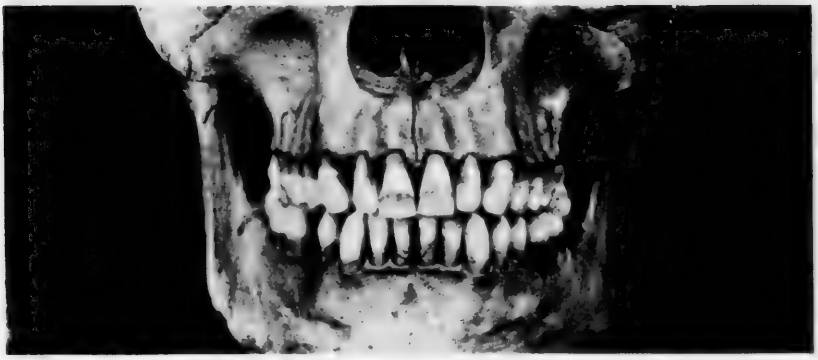

Fig. 25. New Hebridean; Class II

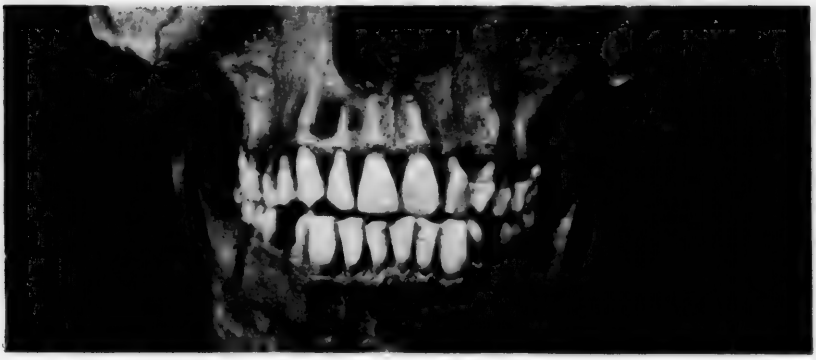

Fig. 26. New Hebridean; Class III

Figs. 24–26. New Hebrideans; Classes I, II, and III.

As we should expect, once the three typal forms of teeth have been demonstrated in all races, these *typal* forms are somewhat more evident and more frequently seen in primitive than in the more modern and amalgamated races. In the enlarged views, *figs. 11*, *12*, and *13*, the three typal forms are very clearly shown.

In *fig. 14*, showing three skulls of native Australians, another very primitive people, the left specimen shows a slightly modified Class I incisor. The other two are very fine specimens of Class II and Class III. In all of the enlarged views the same order of placement is followed as in the preceding illustration, that is, Class I at the top, Class II in the middle, Class III at the bottom. (See enlarged *figs. 15*, *16*, and *17*.)

In *fig. 18* we have a particularly interesting exhibit—three Egyptian skulls of the third and fourth dynasties, a period dating back more than two thousand years before Moses led the Children of Israel out of Egypt. We are back in the Bronze Age at the dawn of civilization, the time of the Troy of the Iliad and before the Great Pyramid was built. In the enlarged views, *figs. 19*, *20*, *21*, we have in Class I and Class III fine typal specimens, while Class II is shown in a slightly modified form.

In *fig. 22* are shown skulls of eight distinct peoples. These skulls vary considerably in size and shape, but the teeth are all of Class III, some of them purely typal in form and others slightly modified. The peoples represented, beginning at the left top, are: Australian, Sandwich Islander, Ancient Egyptian, Kaffir, Chinese, African, New Hebridean, Hindoo.

Figure 23 shows nine specimens of Class I type. Beginning at the top and reading from left to right, they represent: Spanish, Sandwich Islander, New Hebridean, German, Javanese, Hindoo, Fiji Islander, Italian, Ancient Egyptian. All peoples, ancient and modern, have three types of teeth. I find the two plates showing *figs. 24*, *25*, *26*, *27*, *28*, and *29* especially interesting, as in addition to presenting the three types of teeth in two widely different races, one of them savage and the other modern civilized, they show a more refined form of tooth in the savage race. Here again is material for scientific speculation outside the scope and object of this paper.

Finding these three types of teeth in the most primitive and

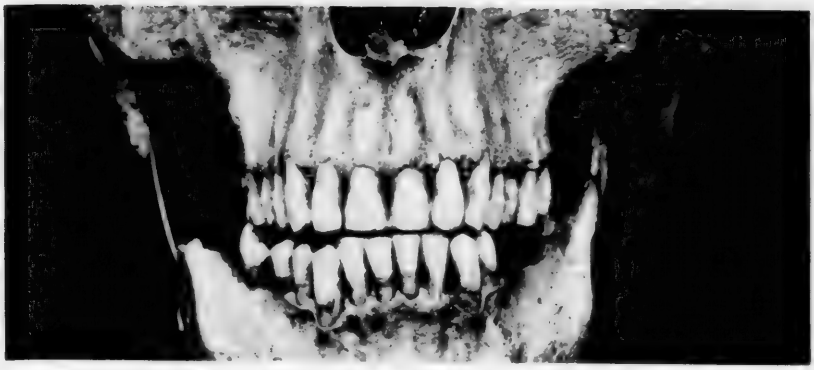

Fig. 27. Modern Italian; Class I

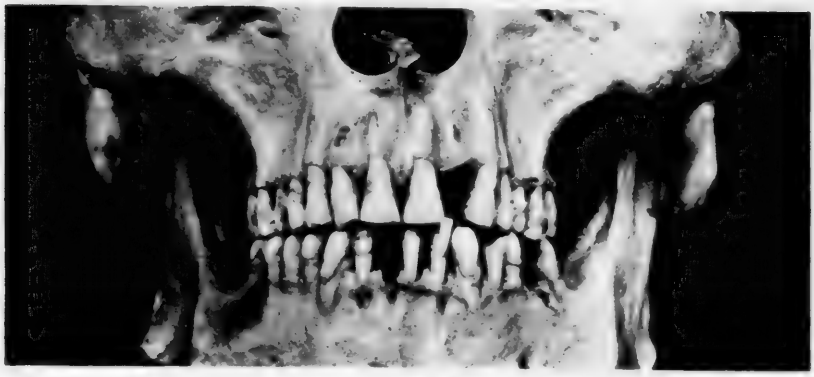

Fig. 28. Modern Italian; Class II

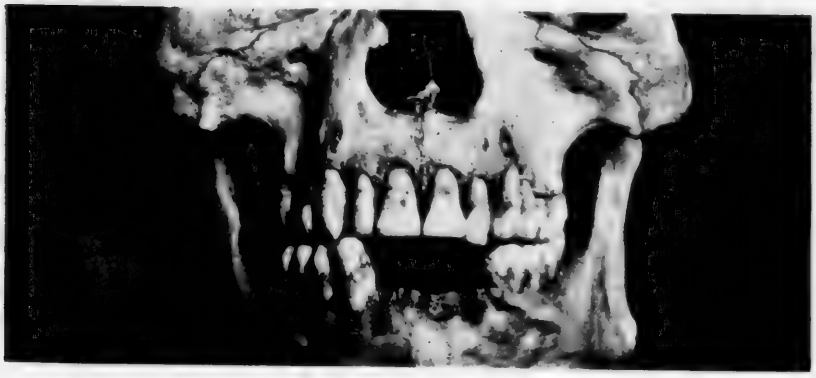

Fig. 29. Modern Italian; Class III

Figs. 27–29. Modern Italians; Classes I, II, and III

ancient races, it naturally occurred to me to make a careful examination of the incisors of the anthropoid apes. Many works have been written on the anthropoid apes, and some of them have called attention to the form of the upper central incisor as characteristic of the ape referred to, but no one, so far as I know, has ever observed that there are the same three types of teeth in the three great apes that we find in man.[1] And these three types of upper central incisors are, as we might expect, even more strongly marked and more clearly differentiated than they are in man. As the nine skulls shown here were selected from less than forty specimens, it is really astonishing that

Fig. 30. Orang-utan; Classes I, II, and III. See *figs. 33, 34,* and *35*

this important fact of three distinct types of teeth in all three of the great apes should have been overlooked so long.

In the material at my command when these studies were made it was only in the orang-utan (*fig. 30*) that the three types were perfectly represented. In the gorilla (*fig. 31*) Classes I and III are perfectly shown; Class II is represented by a modified form. In the chimpanzee skulls (*fig. 32*) Classes I and II are finely represented, while Class III

[1] Just as these lines are being written Dr. W. K. Gregory, of the American Museum of Natural History, calls my attention to a German work by Dr. Von Adolf Remane, "Beiträge zur Morphologie des Anthropoidengebisses," in which drawings showing variations in the forms of anthropoid incisors are presented. But I find no hint that these variations constitute three distinct types nor are these three forms distinctly shown.

is a modification. Enlarged views of the above: *figs. 33, 34, 35—36, 37, 38—39, 40, and 41.*

We see that the previously held views that there is some particular form of tooth characteristic of the anthropoid apes has no foundation

Fig. 31. Gorilla; Classes I, II, and III. See *figs.* 36, *37*, and *38*

Fig. 32. Chimpanzee; Classes I, II, and III. See *figs.* 39, *40*, and *41*

whatever in fact. They have the three types of teeth that we find in all human races, with even more strongly marked or bold characteristics.

In addition to this very striking homology between the teeth of man

and the teeth of the apes in all the major features that I have pointed out, there is another curious resemblance worthy of note. Referring to the first illustration in this paper, you will observe that there is a far greater variation in the teeth of Class I in man, in the proportion of width to length, than in either of the other two classes. This is much more clearly seen in the outline drawing (*fig. 42*) which is accurately made from natural teeth. Owing to a mistake in placement, the order of the Classes is reversed. If you will now look at the central upper incisor teeth as shown in the three skulls on the left of *figs. 30, 31* and *32*, representing Class I in these apes, you will observe the same variation in the proportion of width to length that we find in man. It is singular that this marked variation should be found only in Class I in both man and the apes. Mindful of the fact that it is advisable to present different aspects of a new idea, repeating the salient feature or features with variations, I call special attention to the last plates in this series containing nine photographs, *figs. 43, 44, 45–46, 47, 48–49, 50, 51*. These show considerably enlarged photographs of different races of men and the anthropoid apes.

In the first column we see, beginning at the top, Ancient Egyptian, Tasmanian, Orang Utan, all showing the Class I type of tooth. In the second column we have Tasmanian, Modern Italian, and Chimpanzee, all strongly marked teeth of the Class II type. In the third column, Kaffir, Sandwich Islander, and Gorilla, all three exhibiting beautiful typal specimens of Class III type of teeth. In the gorilla photograph the left central was lost as may be seen by referring to the smaller photograph of this same specimen shown in *fig. 38*. Over the black space in the enlarged print I have pasted a photograph of a left central incisor taken from the Sandwich Islander shown immediately above that of the Gorilla. Both in size and outline the variation is far less than in many human teeth of the same class, ***and no greater than in the centrals in the illustration immediately above it from which it was taken.*** And this is not an isolated instance. I have other specimens in which the resemblance is as close. And while we should avoid laying undue stress on the significance of one or a few specimens, we should also keep in mind the fact that in no other feature of the skeleton of the Gorilla or the other apes has such an astonishing homology been discovered.

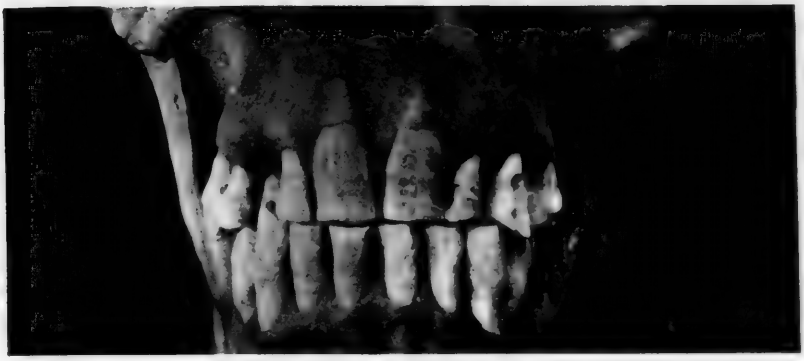

Fig. 33. Orang-utan; Class I

Fig. 34. Orang-utan; Class II

Fig. 35. Orang-utan; Class III. The perspective in this illustration is not quite right. The top of the skull was tipped too far back, thus foreshortening the teeth. But the incisors are a perfect specimen of Class III.

Figs. 33, 34, 35. Orang-utan; Classes I, II, and III. Enlargements of *fig. 30*

But, let me repeat, the convincing evidence of blood relationship between man and ape lies not in a few isolated instances, even when resemblance amounts almost to indentity, but in the universal presence of the three types of teeth in men and apes.

I have said that the mistakes and misinterpretations which have been made in the past with reference to racial or typal forms of teeth have obviously been due to lack of intensive critical study and the examination of too few specimens. A striking illustration of this is seen in a table published as part of a report on the racial characteristics of the labial surfaces of incisors by Messrs. Azoulay and Regnault, and reproduced in Dr. William K. Gregory's monumental and exhaustive work, "The Origin and Evolution of the Human Dentition." These authors, Dr. Gregory says, "measured the difference between the length of the lower border of the central upper incisor and the length of a wire placed parallel to its upper border. The following table gives the results of these measurements:

RACES	NUMBER OF SPECIMENS	DIFFERENCE
Apes (chimpanzees and gorillas, male and female)	5	3.04
Various negroes	41	2.39
New Caledonians, and New Hebrides	13	2.19
Australians	10	2.00
Polynesians	14	2.01
Annamese and Chinese	13	1.78
Europeans	18	1.61
Hindoos, Bengalis	7	1.27

"From this they conclude that the inferior races (Negroes, Papuans) have incisors which approach those of Apes (in being wide at the bottom and narrow at the top). The 'yellows,' the Europeans and the Hindoos, on the contrary, have teeth with parallel borders."

In view of the evidence which I have presented of the three types of teeth in all races and in the anthropoid apes it is difficult to understand how such a mistake could have been made by the authors above quoted. They were, obviously, measuring teeth of Class II in all but the three last-mentioned races, in which they chanced on modified forms of Class I. However, mistakes of this character with reference to the various forms of human teeth and the relationship of peculiari-

Fig. 36. Gorilla; Class I

Fig. 37. Gorilla; blending of Classes I and II

Fig. 38. Gorilla; Class III. Perfect typal specimen

Figs. 36, 37, and 38. Gorilla; Classes I, II, and III. Enlargements of *fig. 31*

ties of form to race and temperament are not uncommon. For half a century it was taught in college textbooks that certain forms of teeth were peculiar to certain temperaments. There was not the slightest foundation in fact for this teaching, as I have abundantly shown in previously published papers. Nearly all of those special features and structures which comparative anatomists have pointed out as common to man and the apes, and of such close resemblance as to give strong grounds for belief in a common origin, are so hidden away in the body that only experts in anatomy have first-hand knowledge of them or can understand their significance.

But here, in the three types of teeth common to man and the apes, we have a feature as open to the perception and understanding of the non-expert as to the trained scientist. The layman will not appreciate the full significance of what he sees, but he will understand enough to give the opponents of evolution an exceedingly difficult task to explain this new evidence away.

To refer once more to the significance of this discovery of three types of teeth in its bearing on the relationship of man to the anthropoid apes, the following letter from the late Professor Ernst Haeckel will be of interest. I had sent Professor Haeckel a package of photographs, including those shown in this article, and this drew from him a prompt response, which reads as follows:

Jena 6, 1, 1913

Dr. Leon Williams,
London.

Dear Sir:—Your observations on the three different types of the upper central incisors are very interesting, and mainly the fact that the same three characteristic types occur also in the orang-utan and in other anthropoid apes. In my opinion this fact is another new and convincing proof for the near relationship between man and the anthropoid apes, and for the phylogenetic theories that both have been derived from one and the same common ancestor. First, the fact that these three types—in physiological relationship of little value—are so distinctly developed in three morphological directions seems to me an important proof that the way of phylogenetic divergence of characters is the same in man and in the anthropoid apes.

Very respectfully yours,
(Signed) ERNST HAECKEL

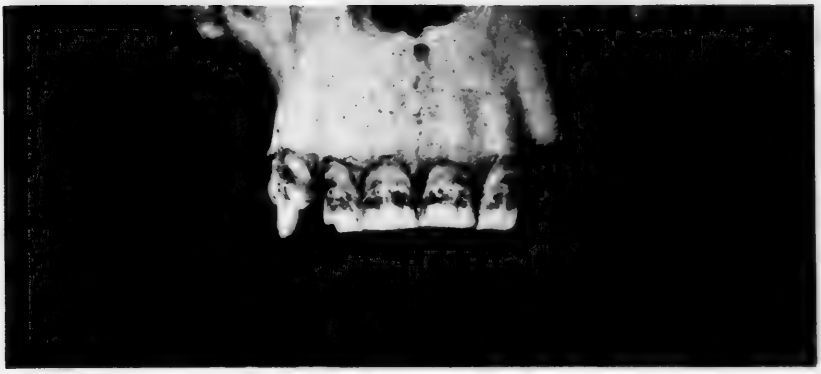

Fig. 39. Chimpanzee; Class I

Fig. 40. Chimpanzee; Class II. Perfect typal specimen

Fig. 41. Chimpanzee; Class III. Slightly modified and worn

Figs. 39, 40, and 41. Chimpanzee; Classes I, II, and III. Enlargements of *fig. 32*

With what admirable clearness and conciseness Professor Haeckel has gone straight to the mark in that letter! Three forms or types of teeth have no special physiological significance, as he says. ***But the morphological meaning conveys the story of man's origin*** and so becomes one of the most fundamental facts about human teeth.

In the face of this well-nigh conclusive evidence of man's near relationship to the anthropoid apes, added to previous evidence that was already overwhelming,[2] what does Professor Osborn have to offer in support of his new view of man's non-relationship to the great apes? First, that man was man and not ape several millions of years ago;

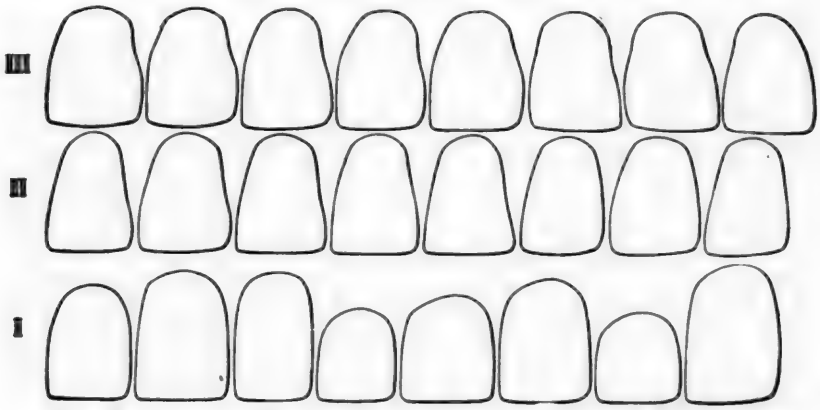

Fig. 42. Accurate drawings from human upper central incisors; slightly enlarged. The Roman numerals at the left indicate the classes.

second, that the anatomical characteristics of the hands and the feet of man do not favor the view that he has descended from arboreal ances-

[2] Those who wish to acquaint themselves with the last word in the whole field of evidence concerning man's relationship to the anthropoid apes should read the masterly presidential address delivered by Prof. Sir Arthur Keith, before the British Association for the Advancement of Science, at Leeds, England, in 1927. Also his book, "Concerning Man's Origin," published by Putnam & Sons. Sir Arthur Keith is generally acknowledged to be the greatest living authority on this subject. In a letter received from him just as this article goes to press he writes: "As to man's relationship to the great anthropoids: one cannot explain man's structure except by supposing he has descended from the stock which also gave origin to gorilla, chimpanzee, and orang. In no other way can we account for the extraordinary number of points which man shares with this animal." This opinion is also that of Prof. William K. Gregory, whose conclusions are based on the most intensive researches extending over many years.

tors. These two propositions seem to me to sum up the gist of Professor Osborn's new views.

In reply to the first, I think we may very properly ask in what way prolonging the vision backward of man's first appearance on earth removes him from relationship to apelike ancestors. What difference does it make to the theory of relationship whether man as man first appeared two million or ten million years ago, if apelike creatures appeared still earlier? If, as Dr. Gregory so cogently points out, man has been on earth for some millions of years, this merely means "that the transitional stages between ape and man must be sought in an earlier geologic epoch." Have not apelike fossil skulls been discovered which are much older than any human fossils? As we proceed backward in the age of these human fossil skulls, do they not, *in some feature or other*, always become more and more apelike? Is not the Heidelberg jaw more apelike than that of the Crô-Magnon man, who appeared much later? And is not the mandible of the still older Piltdown skull more apelike than the Heidelberg jaw? What does this mean if not that man and gorilla resemble each other, or some common ancestor, more and more the farther back we go in the prehistory of both? Again, does the fact, if it be a fact, that man as man appeared on earth several million years ago, with no definite suggestion of an ancestor, contain any hint of an idea that man appeared suddenly, or that he sprang, *per saltum*, from a single pair of ancestors?

Was there any definite period in time or any definite spot on earth when and where it can be confidently stated that man appeared? If not, then must there not be a vast number of regressive forms connecting man with, let us say, the early vertebrates? Surely, the earliest Dawn Man must have had some sort of ancestor.

If the forms immediately preceding man were not apelike, what were they like? Many paleontologists hold that there is overwhelming evidence that man's evolutionary ancestors were apelike animals. On the other hand, I know of no shadow of evidence that man's immediate prehuman ancestors resembled wombats or kangaroos or any other animal known to science.

In truth, I cannot see that Professor Osborn's latest views deal with the problem of origins at all. They seem to me to deal entirely, so far as the facts presented are concerned, with the great periods of

Fig. 43. Ancient Egyptian; Class I

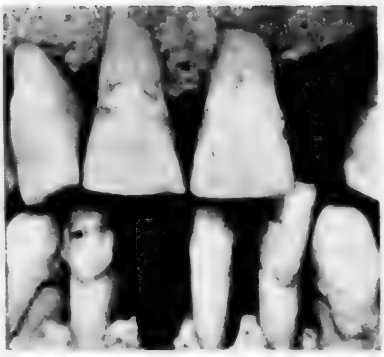

Fig. 46. Modern Italian; Class II

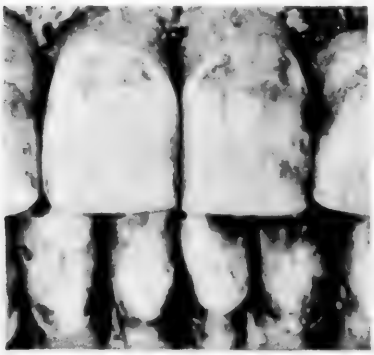

Fig. 44. Tasmanian; Class I

Fig. 47. Tasmanian; Class II

Fig. 45. Orang-utan; Class I

Fig. 48. Chimpanzee; Class II

Figs. 43–48. Different races of men, and anthropoid apes; Classes I and II

Fig. 49. Kaffir; Class III

Fig. 50. Sandwich Islander; Class III

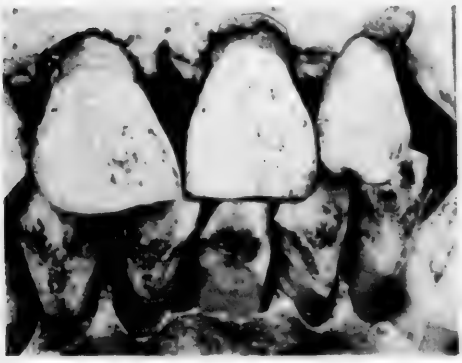

Fig. 51. Gorilla; Class III

Figs. 49–51. Different races of men, and an anthropoid ape; Class III

time covered by man's prehistory. He places the Dawn Man far back in an indefinite prehistoric time and leaves him there without an ancestor of any sort.

With regard to the second point in the summing up of Professor Osborn's argument—that the anatomical characteristics of the hands and the feet of man do not favor the view that he has descended from arboreal ancestors—that position, so it seems to me, has been so effectively disposed of by Professor William K. Gregory that but little more need be said on that subject. I may, however, add one or two suggestions to Professor Gregory's able and convincing arguments.

A million or possibly two million years would seem to be a quite sufficient length of time to bring about such anatomical changes as we find in the human foot when we compare it with that of the gorilla. Are not the changes which we know have occurred in, for instance, the human mandible in half a million years, really greater than the required changes in the foot of the gorilla to give it the form we find in prehistoric man or in the primitive races of today? Furthermore, is it not highly probable that the continued specialization of the gorilla's foot for a million years or more, has greatly increased the difference between the gorilla and the human foot of today? There may have been divergence in two directions.

Finally, I cannot refrain from calling attention to the appealing form of language used by Professor Osborn in a striking statement in his recent book, "Man Rises to Parnassus," as being much more emotional than scientific. The passage to which I refer reads as follows: "Let us befriend the Dawn Man of the long, pre-Stone age and remove from his reputation the bar sinister of ape descent. The myth of our ape-ancestry lingers on the stage, in the movies, in certain anti-naturalistic literature, in caricatures of our pedigree, even in certain scientific parlance, but the ape-ancestry is becoming out of date and its place is being taken by the recent demonstration that we are descended from Dawn Men, not from 'Ape-Men.'"

That statement will gladden the hearts of all the orthodox; it will bring joy to every Fundamentalist who reads it. It is no calmly reasoned sequence of thought based on fact, on what we know, but an appeal to a deep-seated prejudice. I have an idea that the psychological basis of this prejudice has never been fairly and squarely stated.

Why should we have a feeling of repugnance for the thought that we have a blood-kinship with the ape, but no such feeling with reference to any other animal? Is it not because we do not resemble any other animal so much as we resemble the ape? Is not the repugnance due not so much to the thought that the ape resembles *us* as to the thought

Fig. 52. A Tasmanian man, showing a very low type of face, with an excessively wide flat nose. Courtesy of Sir Harry Johnston. Photograph by J. W. Beattie, Hobart, Tasmania.

and the evidence that *we*, in more ways than one, resemble the ape? And do we not have something of the same feeling of repugnance when we look at any degraded and loathsome human being?

From the general drift of all the objections to the Darwinian theory of man's relationship to the ape, I gather that if the gorilla were only a little more unlike man or farther away from man in the ancestral

line, the idea would probably arouse little or no antagonism. But we despise the poor and inferior relation whose kinship is near and known.

Look at this photograph of the last of the Tasmanians (*fig. 52*). You cannot deny your blood-kinship with that creature, but does not the sight of this face, so much more closely resembling the face of a gorilla than does your own, arouse in you a strong feeling of repugnance? But all evidence we have shows that the faces of the Dawn Men were strikingly like this face of the last of the Tasmanians although probably even more gorilla-like. Here is a "bar sinister" that we cannot remove. What about it? Does not the "bar sinister" apply as strongly to *Dryopithecus*, or any other ape-like creature, as to the gorilla?

Pride in ancestry is no doubt a very deep-seated human feeling, particularly if we may call attention to some reflected glory shining upon ourselves. Many ancient peoples, primitive and highly civilized, and many individuals have claimed or had claimed for them descent from the gods or from supernatural beings. But I suppose such a claim would not be seriously discussed today, not even for the very superior Nordic race.

Professor Osborn says that only an ape-mind can spring from the ape. Is not that statement, in its implications, completely destructive of the whole theory of the descent of higher from lower forms of life? If something higher than the ape may not spring from the ape, what warrant is left for the belief that any higher form of life may arise from a lower form? But if we slightly change the relationship of the ideas in Professor Osborn's proposition, it seems to involve him in still further difficulties.

Did the Dawn Man have none of the qualities and proclivities of the ape or the tiger? If not, we are well on our way back to the Garden of Eden story, or we should be if we did not find those same qualities and proclivities in many specimens of the human race of today. If we did not inherit them from the Dawn Man, and through him from the ape, where did we get them? There is more than a little to be said in defense of the proposition that man's descent from lower forms of life is even more evident in his mind than in his body.

But we have come far, very far, from the ape and the Dawn Man, and therein lies our great hope for the future of humanity, for a race of

supermen. Races, peoples, nations, civilizations may rise and fall, come and go, but humanity itself seems to have always been moving forward. And the great ones, the poets and the prophets, seem always to have had their eyes fixed upon some far-off high destiny. In man's lowly origin we find the pledge and promise of his future. He is not descended from gods, but he may be on the way toward becoming godlike. And the ape-stage marks the period when he first began to look upward.

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